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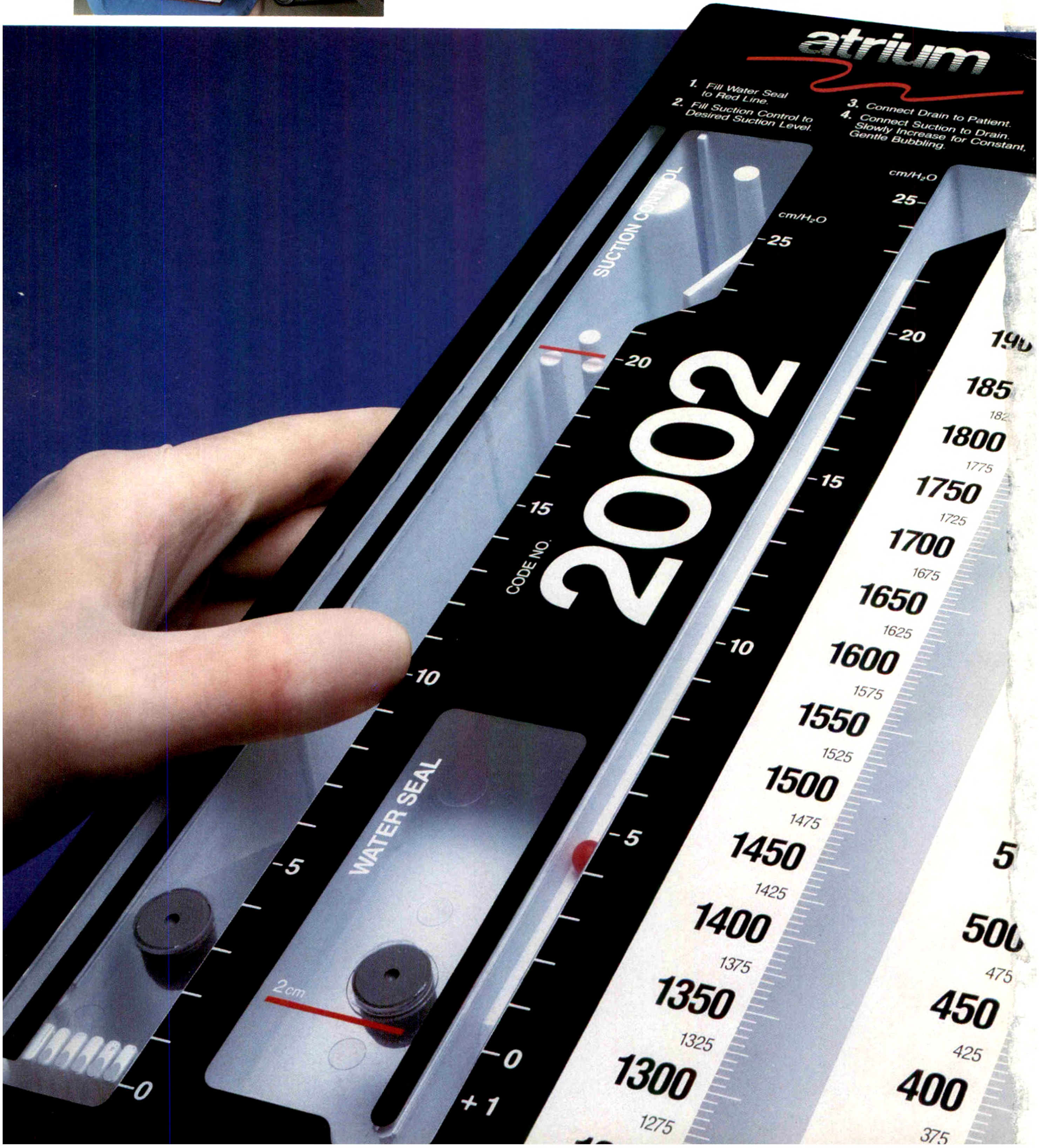
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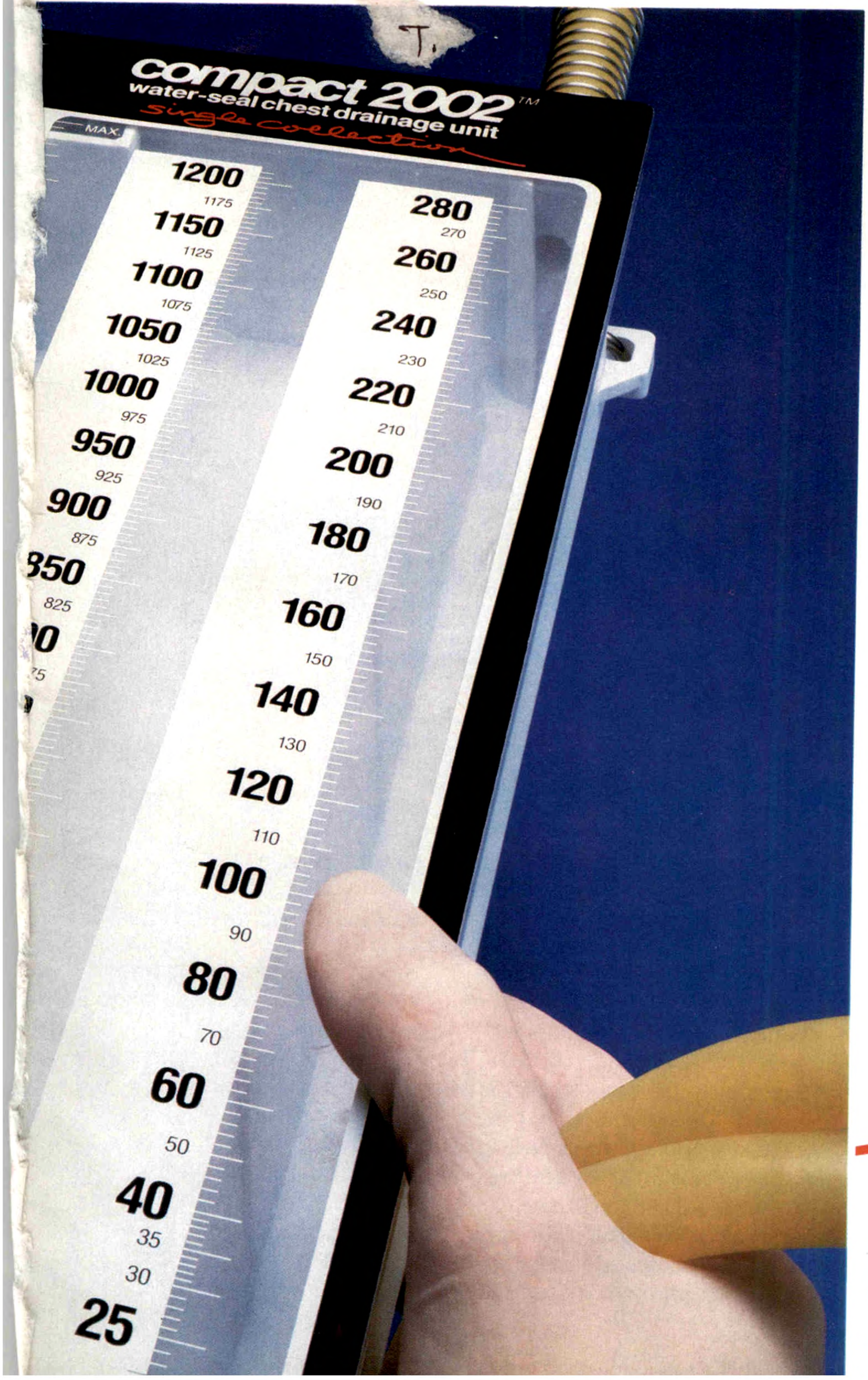

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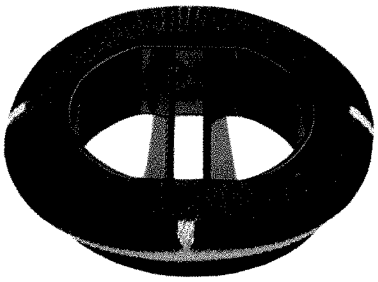

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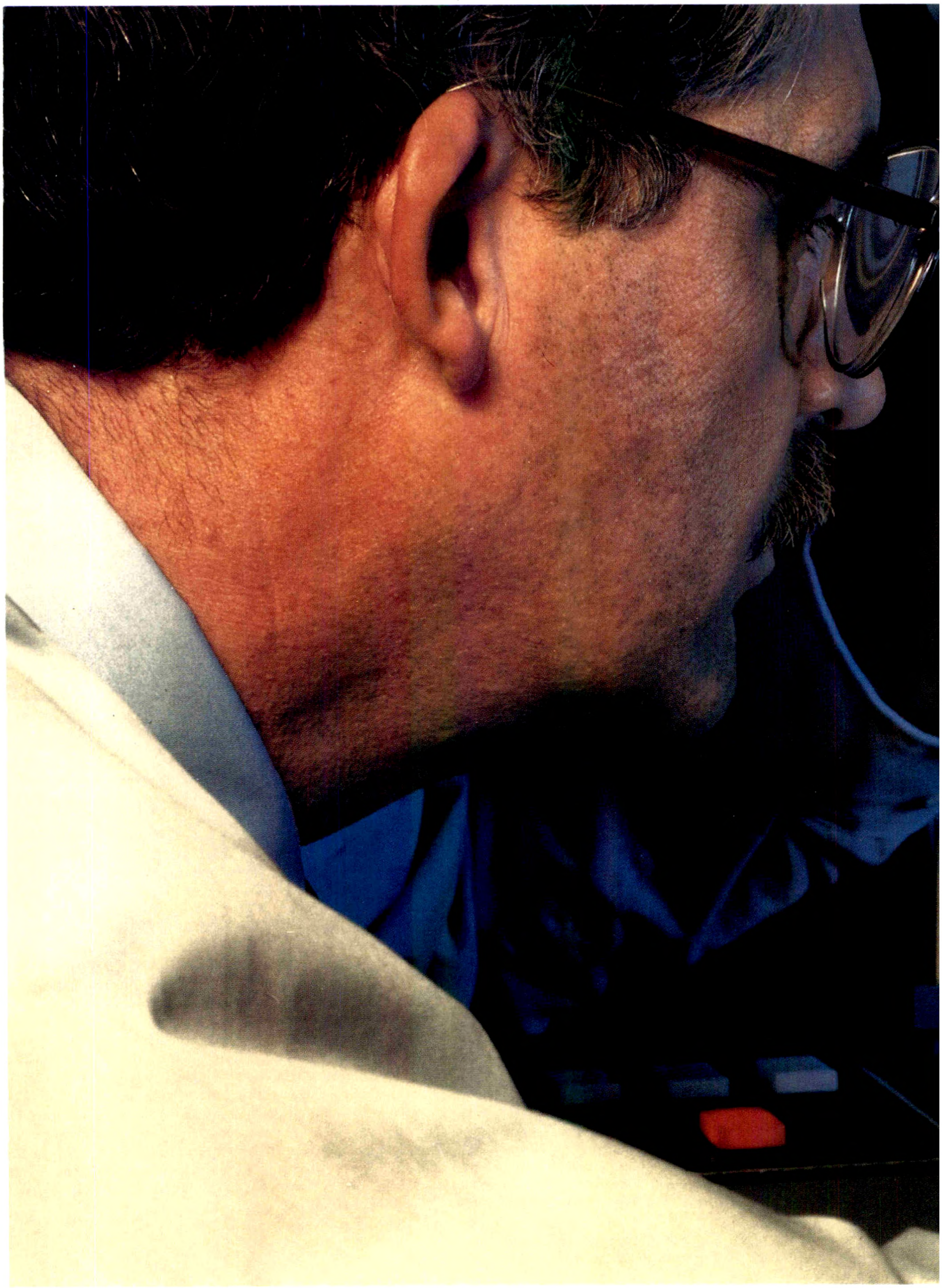

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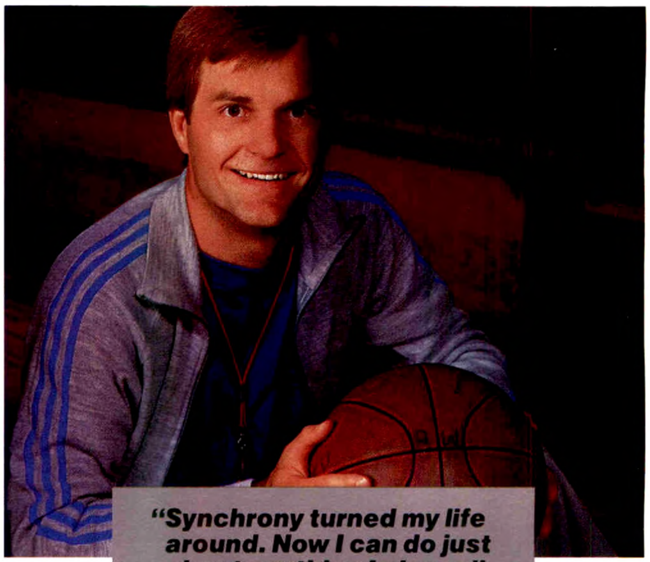

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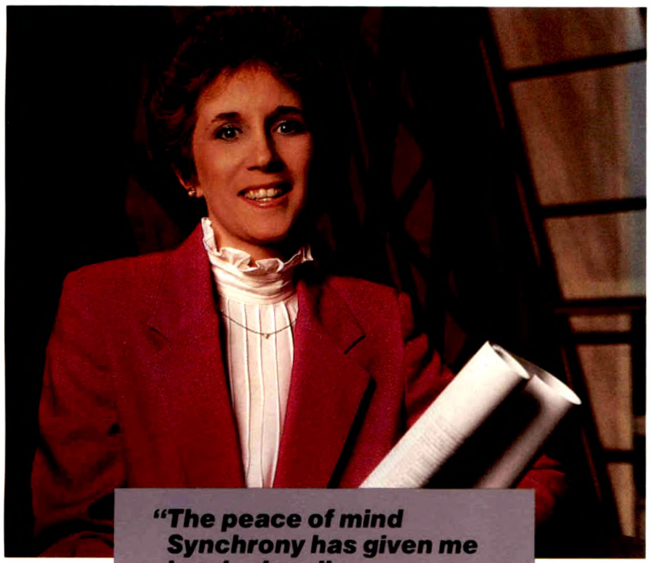

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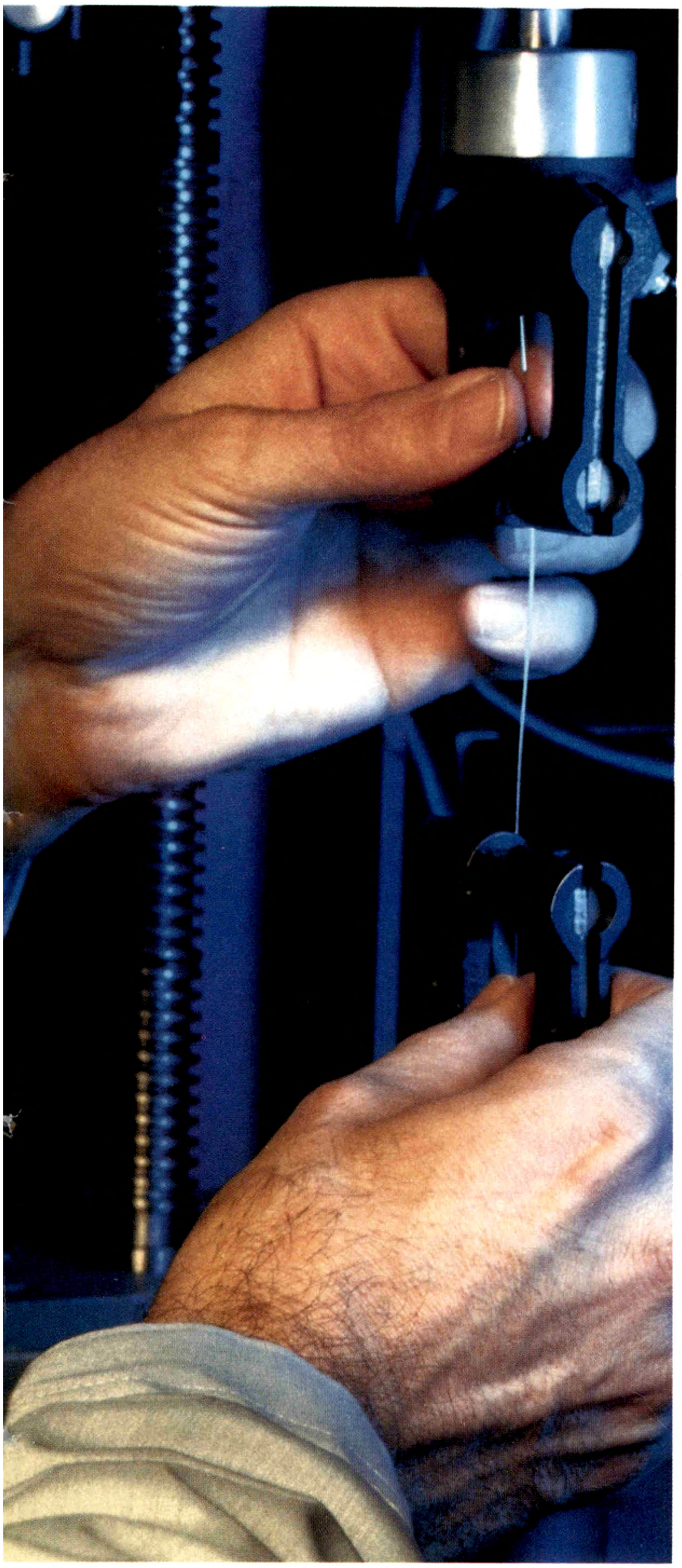

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Tissue Adhesive in Bronchial Closure
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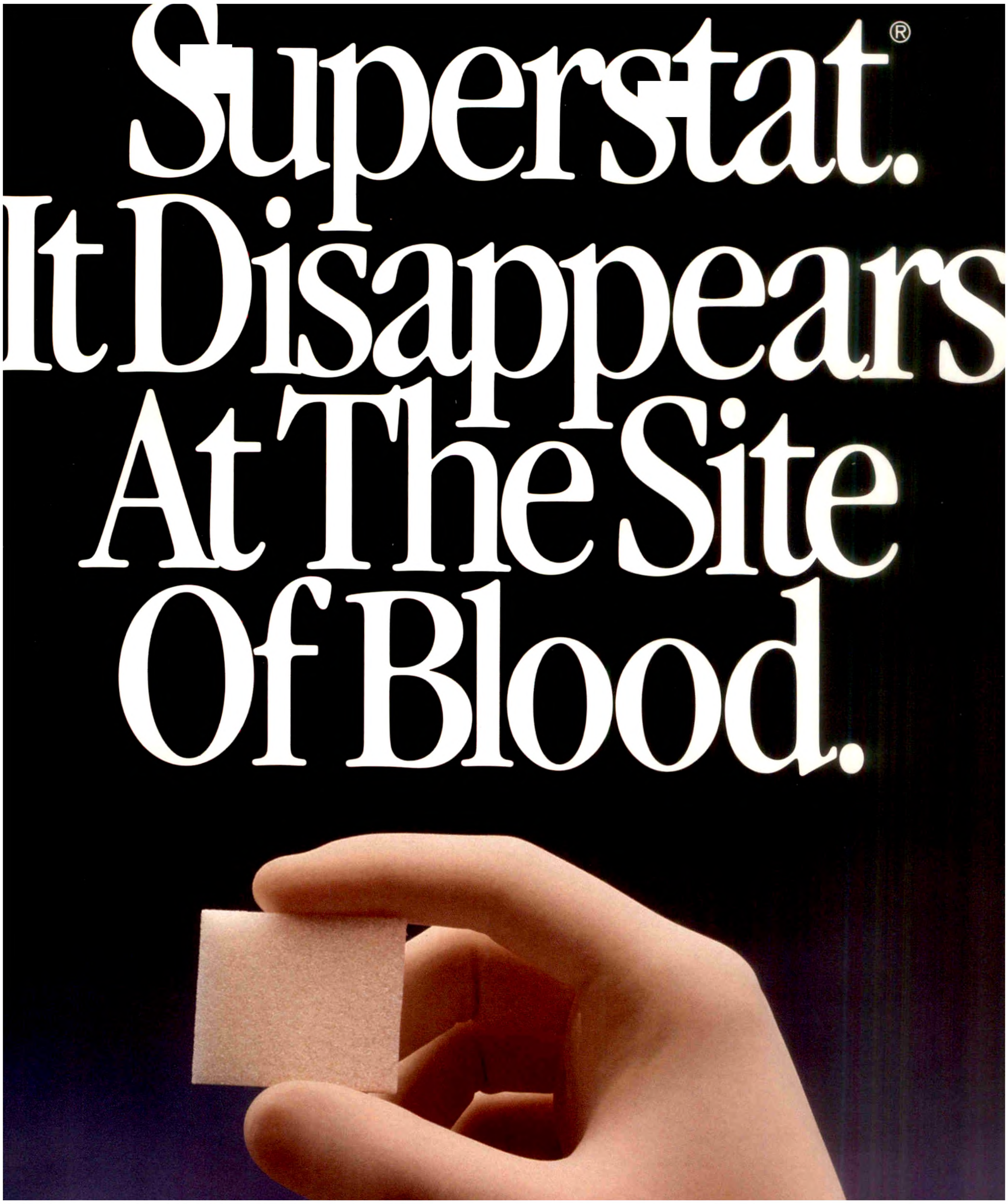
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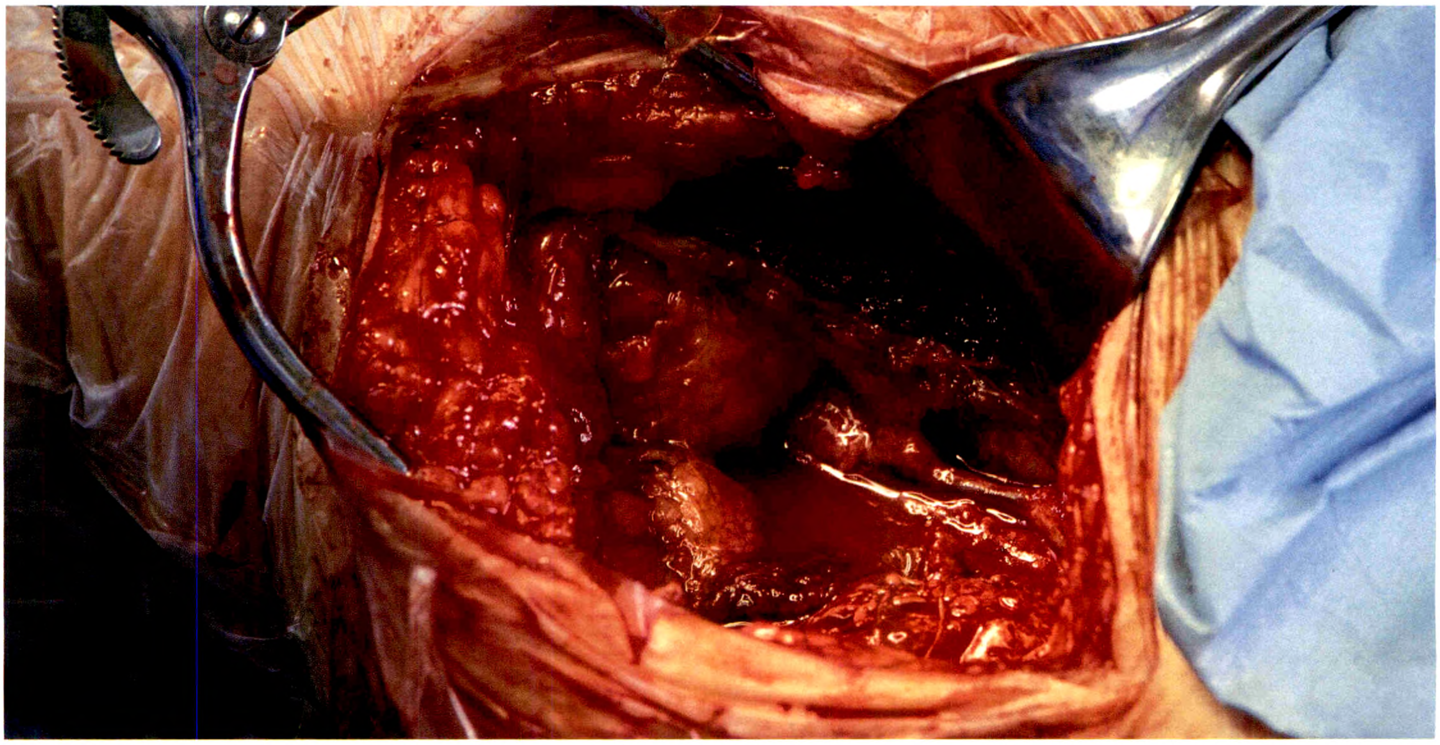

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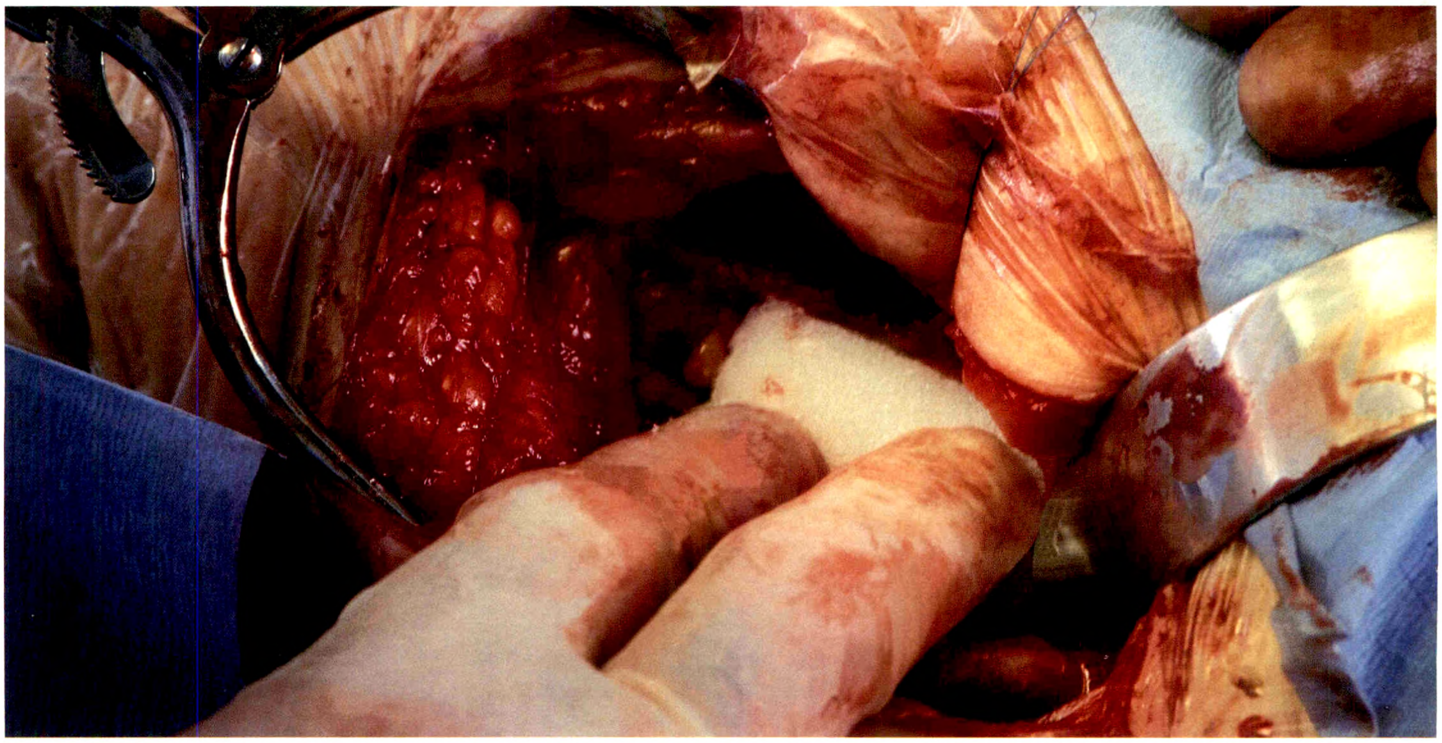

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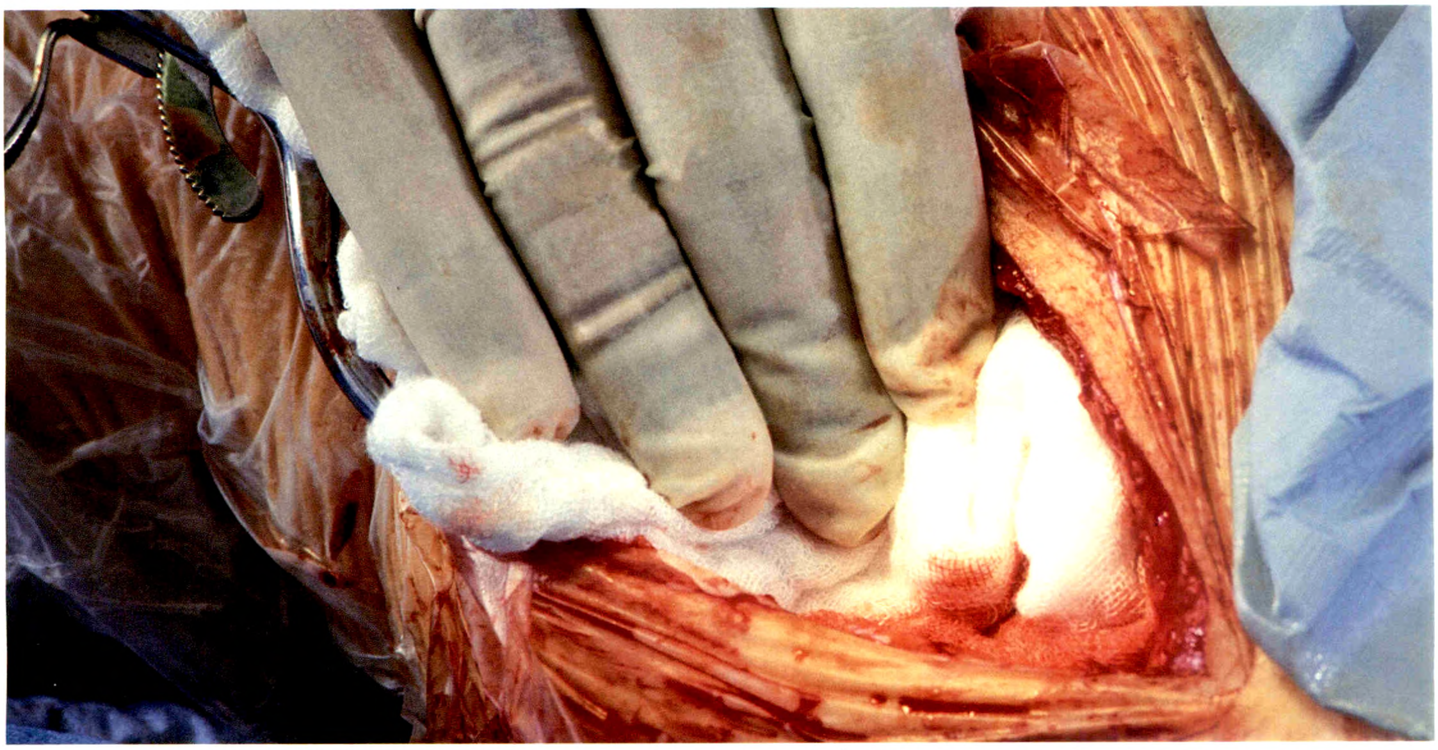

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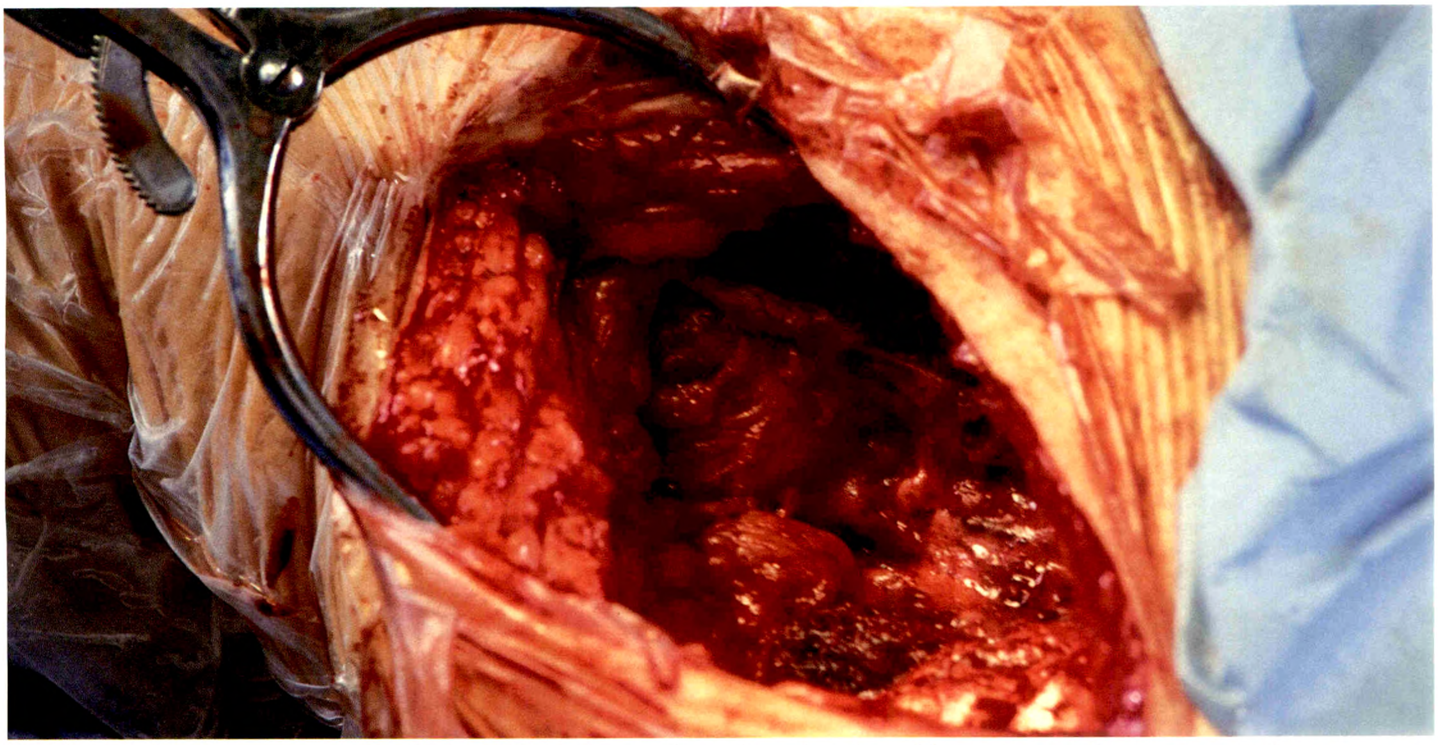

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Treatment of Malignant Tracheobronchial Obstruction

Richard B. McElvein, MD

Division of Cardiothoracic Surgery, Department of Surgery, The University of Alabama at Birmingham, Birmingham, Alabama

Tumors of the trachea and main bronchi may be primary, a manifestation of pulmonary carcinoma, or metastases from other sites, notably thyroid, breast, and kidney. Patients with symptomatic malignant airway obstruction have been and remain a difficult problem for surgeons. As might be anticipated, a variety of methods of treatment have been used in the past on an elective, urgent, and in a few cases, emergent basis. Historically, tracheostomy was used and proved effective for obtaining an airway if the tracheostomy tube could be placed below the level of obstruction. A variety of other intratracheal or bronchial stents have been devised including Montgomery T tubes, modifications of tracheostomy tubes, and even a bifurcated silicone rubber stent that lies in both the trachea and main bronchi [1, 2].

Destruction of an airway tumor has been accomplished by the use of cryotherapy and endobronchial electrocautery [3, 4]. Definitive resection of primary neoplasms of the trachea and carina can be carried out with moderately good results providing the lesion can be completely eradicated [5]. More recently laser therapy with either the carbon dioxide laser or the neodymium:yttrium-aluminum-garnet (Nd:YAG) laser has proved to be a safe, effective method of caring for these patients [6, 7].

Palliation can also be achieved for patients with recurrent endobronchial malignancy by the use of high-dose intraluminal irradiation using a radioactive source remotely after loading it into a catheter that has been bronchoscopically placed in the airway [8]. Sometimes heroic measures are called for, as evidenced by a patient with a tracheal cylindroma who, while undergoing radiation therapy, developed a critical tracheal obstruction for which she was placed on extracorporeal circulation and underwent bronchoscopy and placement of a stent [9].

In this issue, Mathisen and Grillo [10] describe their experience using the rigid bronchoscope to core out a tumor to provide an improved airway with subsequent management by radiation therapy or resection, or both. In their description of the technique, they identify many of the elements that are common in all of the methods that have been used. Many of these patients are seen with a critical illness, and the diagnosis of major airway obstruction is not in doubt. What is in doubt and what must be ascertained is the site and extent of the obstruction so that an appropriate treatment method can be chosen. Standard chest roentgenograms and tomograms of the airway are far superior to computed tomographic scans of the thorax in obtaining this information. The management of these patients requires close cooperation between a skilled anesthesiologist and an experienced bronchoscopist with all methods of ventilating a patient being available, including standard endotracheal tubes of varying sizes, ventilating systems that will attach to rigid bronchoscopes, and catheter ventilation systems. The availability of a peripheral pulse oximeter makes management of these patients much easier, as oxygenation can be monitored continuously and if hypoxia occurs additional measures can be carried out to improve oxygenation.

Mathisen and Grillo make the point that the rigid bronchoscope can be used effectively to core out an obstructing airway lesion with limited bleeding and an excellent chance of providing an improved airway. Once an adequate airway has been established, it is then possible to evaluate the patient more thoroughly and plan on definitive treatment as appropriate. A serious defect of this method is the limited ability to treat distal main bronchus and lobar obstructions, both of which can be treated effectively with lasers.

The procedure of choice in managing malignant airway obstruction is evolving. The skill and experience of the endoscopist and available equipment may be determining factors in choice of a method. Mathisen and Grillo have described one method of managing malignant airway obstruction; there are others.

A review of the last 50 consecutive cases of malignant airway obstruction treated by my colleagues and me from March 1987 to the present with the Nd:YAG laser (36 patients), a carbon dioxide laser (4), or combined Nd:YAG and carbon dioxide lasers (10) revealed substantial advantages to our method.

Thirty-six male and 14 female patients were treated from one to more than five times. Twenty-eight patients (56%) were treated once, but 22 patients (44%) were treated more than once for continuing control of their disease. Our experience was analyzed to determine which patients could have been managed by the technique of Mathisen and Grillo. Of the 50 patients, 8 could have been treated in this fashion. Fifteen of the patients might have been treated with the coring-out technique but were better served by the use of a laser. Twenty-seven patients could not have been managed without the use of the laser because their lesions were in the distal airway or obstructing an upper lobe.

This review of our patients reinforced our decision to abandon the technique described by Mathisen and Grillo. We conclude that the technique of rigid and flexible bronchoscopy in conjunction with both the carbon dioxide and Nd:YAG lasers provides more flexibility in managing a larger number of patients with serious respiratory tract obstruction and should be considered the method of choice.

Address reprint requests to Dr McElvein, Division of Cardiothoracic Surgery, Department of Surgery, The University of Alabama at Birmingham, University Station, Birmingham, AL 35294.

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Crossovers in Coronary Artery Bypass Grafting Trials: Desirable, Undesirable, or Both?

Lloyd D. Fisher, PhD, George C. Kaiser, MD, Kathryn B. Davis, PhD, and
Michael B. Mock, MD

Coordinating Center for Collaborative Studies in Coronary Artery Surgery, University of Washington, Seattle, Washington; St. Louis University School of Medicine, St. Louis, Missouri; and Mayo Clinic, Rochester, Minnesota

In three major randomized clinical trials of early coronary artery bypass grafting (CABG) versus medical treatment, substantial numbers of patients were randomized to each treatment arm [1–3]. In this issue of *The Annals,* Weinstein and Levin [4] explore the effect of crossovers from medical treatment to CABG. They discuss the adverse effect on statistical power and the probability of detecting treatment differences that results from crossovers from the medical to the surgical group. Clearly, crossovers can have an adverse effect on power. This is not a new concept; the effects of crossover on power were previously examined in the baseline article of the Coronary Artery Surgery Study (CASS) [5]. Although we agree with Weinstein and Levin that crossover diminishes power, a number of other related issues deserve comment. First and most important, they do not consider the definition of the relevant medical question. Second, they have not used the best method to compute statistical power. Finally, there are inherent biases in their suggested alternatives to the intention-to-treat analysis.

Definition of the Medical Question

Consider two possible randomized trials of CABG for mild angina that have survival as their endpoint. In trial A, patients are randomized to either operation or medicine and are not allowed to cross from medicine to operation. In trial B, patients are randomized to either treatment as initial therapy. If angina develops in a medically assigned patient who is inadequately controlled by medical therapy, the patient is allowed CABG for pain relief. The two trials are designed to answer different questions. Trial A addresses the question: What would be the effect on survival of abolishing CABG as a treatment option? Trial B addresses the question: If we postpone CABG until angina dictates operation and then allow operation, is survival adversely affected? Although trial A addresses an interesting biological question, this narrow result would not be relevant to the treatment of these patients with a changing clinical course. In addition, it would be unethical in our society to deny the patient with severe uncontrolled angina the potential pain relief of CABG. Trial B is relevant to medical practice. Fortunately, trial B is the type of trial that has been performed. The ethical dilemma was recognized in the design stage of CASS; the operations manual included provision for crossover for inadequate medical control of angina. Repeat CABG was also allowed in the surgical group.

The results of trials that allow crossover must be qualified. As Weinstein and Levin observe, patients with increasing symptoms who cross over from medicine to operation may be patients at high risk. Postulation of increased risk for untreated acute ischemia is in accord with clinical observation and biological intuition. In a CASS observational registry study of patients who would have been eligible for the CASS trial except that their angina was too severe, the comparison with medical therapy showed a benefit for surgical therapy [6]. However, the probable benefit of operation for patients with intractable angina does not imply a benefit when operation is performed before patients reach this point. The crossover in the CASS randomized patients may be a response to an acute need for revascularization. For the CASS nonrandomized patients who were eligible for randomization but received medical therapy by physician or patient preference, crossover to operation occurred at the same rate as that of randomized patients [7].

In the interpretation of studies of CABG that allow crossover, it is pertinent to appraise other factors associated with each treatment strategy, such as number of hospitalizations, length of hospitalizations, medication use, patient activity level, and employment [8]. The effects of delayed operation should also be considered. Although many trial patients assigned to medical therapy later received CABG, the majority did not. If operation is deferred for a time, the need for resources to perform the operation is also deferred. Furthermore, a delay in operation will delay the consequences of potential graft deterioration. Repeat CABG and loss of the surgical benefits may be eliminated or delayed. Integration of these measures with survival results is necessary when treatments are compared.

Computation of Statistical Power

Patients initially assigned to medical therapy cross to surgical therapy over time. The model of Weinstein and Levin does not account for the timing of the crossover, yet it is essential to include this in the model. One method of incorporating time of crossover is described in the CASS baseline article [5].

Another important problem lies in the rate assumptions used in the power calculations. Weinstein and Levin assume that the mortality rate in the noncrossovers remaining in the medical group is the same as the mortality

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rate would be in the entire group of medically assigned patients if crossover were not allowed. This is not a reasonable assumption. Suppose there is an acute medical event that occurs in many patients and leads to death in the seriously ill patients. Suppose that for the stronger patients survival after this event is not affected by treatment. If this event (progressive angina) often leads to crossover (operation), the survival in the noncrossover group will be artificially lowered. Because none of the seriously ill patients survive long enough to cross over, the medical group is weighted with all the deaths. In addition, a large number of the low-risk survivors are removed from the noncrossover group. Use of the model of Weinstein and Levin would yield a spuriously high estimate of mortality in the noncrossover group. Weinstein and Levin also assume that the mortality rate in surgically assigned patients is the same for patients assigned to operation and patients who cross to operation. This assumption may be questioned for similar reasons.

Alternatives to Intention-to-Treat Analysis

The intention-to-treat analysis, wherein patients are analyzed in the groups to which they were randomly assigned, preserves the advantages of randomization. This is a very important advantage [9]. In general, the intention-to-treat principle leads to conflicts between statistical goals and biological goals when there is poor design or execution of a study [9]. As Weinstein and Levin acknowledge, an intention-to-treat analysis of the CABG randomized trials can lead to semantic difficulties. In standard usage, a medically assigned patient who undergoes operation is called a "nonsurgical" case. We agree that this distorts the clear meaning of the language. Perhaps terminology should be changed to "early medical treatment group" and "early surgical treatment group."

Weinstein and Levin suggest alternatives to the intention-to-treat principle. The first suggestion is to drop crossovers from the study. If this were done, the result would be a tremendous bias against the medical group, as only patients who survive long enough to be eligible for operation could be removed. Alternatively, patients could be considered medical cases until they cross to operation. In this case the interpretation is more complex and biases in either direction could result. The second suggestion is to include the medical patients in the medical group until the time of crossover and then to move them to the surgical group. Weinstein and Levin argue that the introduction of moribund patients into the surgical group would bias the results against operation, although in the appendix they acknowledge that in the CASS trial the crossovers had a lower mortality rate than the original surgical patients. In fact, this approach could introduce bias in either direction. The last suggestion is to abandon the study. If randomized trials are abandoned, the issues would be decided based on purely observational data, with which the possibilities for bias as well as the analysis difficulties are much greater.

Inability to Distinguish an Effect

In one important area we are in total agreement with Weinstein and Levin. They note that the failure to reject the null hypothesis of no difference does not show that no difference exists between treatments. When no significant difference is found between therapies, one must examine the estimated effect of the different treatment arms. Crossover will decrease the power to detect differences between "pure" treatment strategies, but crossover will not decrease the power to detect differences between treatment strategies when crossover is considered part of the strategy.

Summary

Weinstein and Levin note that the intention-to-treat principle has been justified as presenting a policy of treatment rather than treatment itself; they note this can "readily degenerate into sophistry." For CABG, however, we argue that treatment policies are indeed the issue. Patients with mild angina do progress and need revascularization. Therefore, the question relevant to patient care is whether revascularization can be deferred until the angina progresses. It is not only appropriate to allow crossover, it is necessary to allow it in order to answer this question.

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Are Oxygenators (Airplanes, Oil Spills, Pesticides) Safe?

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Robblee and colleagues [1] report observing the sudden onset of hypoxemia during a resumed cardiopulmonary bypass with a Bentley BEN-10 B bubble oxygenator. When increasing oxygen flow did not improve "color or saturation" and gas release through the pressure release valve was evident, the bypass was discontinued. Subsequent examination of the oxygenator showed crystalline mannitol occluding the oxygen line to the gas inlet port. Robblee and colleagues discuss briefly the roles played by retrograde gas line filling, mannitol concentration, polyvinyl chloride surface interactions, and temperature. They analyze the problem and show the mechanism of accident. They conclude that mannitol should not be added to the prime, unless in dilute solution, until gas flow has been started.

A search of the National Library of Medicine, using the computer modem program GRATEFUL.MED, has not yielded any report of a similar problem. Does this mean that the problem is previously unknown, a rare occurrence, or a unique event, or that the search was inadequate? Not at all. To a considerable extent, the users of cardiopulmonary bypass are flying blind, without full instructions and with inadequate disclosure of problems experienced by other surgeons because detailed information often simply is not available.

Not surprisingly, Robblee and colleagues describe their complication as life-threatening but strongly maintain that such problems are very infrequent and cite the excellent 1978 article of Mortensen [2] in support. This article should be read carefully in its entirety as one of the very few accessible accounts of oxygenator safety and efficacy. After stating that, despite their complexity, the relative freedom from serious problems in use of commercially available disposable oxygenators is "striking and significant," Mortensen [2] continues:

> Hard data describing harmful effects, errors, failures, or problems observed during clinical use of oxygenators, as with any other new mechanical device, are difficult to obtain. This is understandable in view of medicolegal considerations, reluctance to report publicly on poor results, problems, failure, or errors, and the iatrogenic, accidental, inadvertent, or episodic nature of most of the problems observed.

On the basis of extensive review, Mortensen [2] identified 55 different problems known to have occurred with use of disposable oxygenators. These are divided into five categories and are listed tabularly.

I wrote earlier [3]:

> It has been easy to believe that clinical perfusion is highly developed, eminently safe, and requiring only more and better trained perfusion personnel, and perhaps a few minor embellishments to be optimal. Unfortunately, clinical perfusions are still potentially very dangerous. A few moments distraction, the wrong reaction to some new situation, a sudden component malfunction—any such event may trigger a catastrophe. . . .

It should not be surprising then that occasional serious oxygenator problems do occur, but how frequent are they really? In 1979, Stoney and colleagues [4] reported that a survey of 349 cardiac surgeons, comprising patient information in 374,819 operations using a pump oxygenator between 1972 and 1977, showed that "a pump oxygenator accident serious enough to cause patient injury or death occurred once in 1,000 procedures." There were 264 deaths. Curiously, this article, which might have altered the tone of the current report, is not even referenced by Robblee and colleagues [1].

Perhaps the reference was omitted on purpose because it does not deal exclusively with oxygenators and only six "oxygenator failures" are reported. The oxygenator is but one component of a very complex system used in humans with substantial but largely unknown risk. My suggestion to the Food and Drug Administration Cardiovascular Panel Subcommittee on Devices on September 12, 1975, that "oxygenator manufacturers should be required to keep records or logs of observed instances of oxygenator failure, malfunction, or inadequacy [2]" has not, I believe, been implemented.

The manufacturers, seeking to avoid disclosure of performance that would have been required by the oxygenator standards being developed by the American Society for Artificial Internal Organs in the 1970s, undertook a competing standards effort in their Health Industries Manufacturers Association. Subsequently, the standards activities of the American Society of Artificial Internal Organs and the Health Industries Manufacturers Association were joined in the Association for the Advancement of Medical Instrumentation [5]. However, about 1984, after the manufacturers had been unable to have oxygenators reclassified by the Food and Drug Administration, they apparently were less interested in the development of consensus standards and withdrew much of their support. As a result, the Association for the Advancement of Medical Instrumentation discontinued activities toward an American consensus standard for oxygenators, continuing only their part as Secretariat in the international effort.

I believe that the major thoracic societies should evaluate the situation. Although oxygenator manufacturing

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standards (avoidance of nontoxic components, use of clean rooms, and maintenance of production testing and records) are high, it is still very dangerous not to have an oxygenator standard that requires full disclosure of device function. Without this, there is no requirement that manufacturers diligently seek records of all failures, malfunctions, or inadequacies, include a summary of this information with the oxygenator instructions, and supply a full listing on request. Because the perceived medicolegal risks discourage many surgeons from reporting pump oxygenator accidents and failures, disclosure should be mandatory so that the record can be more complete and corrective measures can be encouraged.

The oxygenator is only one component of a potentially very high-risk circuit. Not only is an oxygenator standard needed, but standards for the entire system should be developed. Consensus standards take a long time and much effort. They have advantages, however. A broader involvement of interested parties is possible. Arbitrary, bureaucratic, or dictatorial decisions are less likely to be made than when a single organization (eg, the Food and Drug Administration) or group of related organizations (eg, manufacturers) controls the result. The ultimate solution of this problem is certainly a responsibility of cardiac surgeons.

The lack of oxygenator use information makes it very difficult or impossible to evaluate realistically the value of the article by Robblee and colleagues. Probably this type of gas line obstruction, or a similar accident, has happened before, perhaps many times. Instructions regarding prevention and more complete use cautions should certainly be included in the information supplied with the device. In addition, careful consideration should be given by the manufacturer to preventing reflux of priming solution or blood into the air line by the installation of a suitable one-way valve.

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Endoscopic Relief of Malignant Airway Obstruction

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The ability to manage acute airway obstruction can be life-saving. Airway relief should be expeditious and immediate, with low morbidity and mortality. It should not interfere with future definitive therapy. In patients with terminal malignancy, it should be economical in cost and should minimize hospitalization. We used biopsy forceps and the rigid bronchoscope to "core out" 56 patients with obstructing airway neoplasms. The location of the obstruction was trachea in 16 patients, carina in 24, main bronchi in 8, and distal airway in 8. Improvement in the airway was accomplished in 90% of patients. A single bronchoscopy was sufficient in 96%. Nineteen complications occurred in 11 patients: pneumonia in 5, bleeding in 3, pneumothorax in 2, hypoxia/hypercarbia in 2, arhythmias in 6, and laryngeal edema in 1. There were four deaths within 2 weeks of core-out related to respiratory failure. Further therapy consisted of resection in 28.6% (tracheal in 9, carinal in 3, pulmonary in 4), irradiation alone or in combination with chemotherapy in 60.7%, and no therapy in 10.7%. Palliation of symptoms and establishment of an airway in acute obstruction is the goal. Survival depends on the effectiveness of the proposed treatment. We find this time-honored method superior to use of the laser.

(Ann Thorac Surg 1989;48:469–75)

Stridor, acute respiratory distress, and obstructive pneumonia may be the presenting signs of advanced airway neoplasms. These neoplasms may be rapidly growing or slow-growing. Symptoms depend on location and extent of the tumor. Neoplasms of the trachea and carina may cause obstructive airway symptoms as opposed to neoplasms of the distal airway, which are more likely to show initial symptoms of postobstructive pneumonia. With the bronchoplastic techniques currently available, neoplasms of the trachea, carina, main bronchi, and lobar orifices may be amenable to surgical resection. In patients with such obstructions, it may be necessary to open the airway to allow obstructive pneumonia to clear or to facilitate airway management at the time of operation. Opening an obstructed airway in patients who have acute respiratory distress with potentially resectable tumors will allow time for a thorough workup, correction of underlying medical problems, or tapering of steroid administration if the patients have been treated for "asthma." Many patients with advanced tumors have obstructed airways, and no surgical therapy is available. In such patients, palliative relief of symptoms is necessary to provide a good airway, allow clearing of obstructive pneumonia, and improve quality of life. It may also be necessary to open the airway to allow palliative treatment such as radiation therapy to be performed.

Many methods of opening obstructed airways are available. The ideal method should be simple, expeditous, safe, and readily available to many physicians. It should allow immediate establishment of an airway and should not require multiple procedures. Many of these patients do not tolerate multiple procedures or general anesthetic agents. The procedure should not interfere with proposed resection or other therapy such as irradiation or chemotherapy. It must be cost-effective and time-effective for patients with advanced malignant disease, as long-term survival is not likely. It must be a procedure that can be accomplished with low morbidity and low mortality. We have used the time-honored method of endoscopic removal of tumor to achieve these goals. The combination of biopsy forceps and the tip of the rigid bronchoscope to "core out" the tumor has been very effective in our hands and satisfies many of the requirements for opening an obstructed airway.

Material and Methods

We reviewed the charts of 56 patients who underwent endoscopic removal of tumors of the airway between 1977 and 1988 at Massachusetts General Hospital. This is a consecutive series of patients. During this time period, 2 patients were unsuitable for our technique and were referred for laser therapy.

Technical Considerations

RADIOLOGICAL ASSESSMENT. If time permits, careful radiological assessment is a valuable asset to operative management. Tomography of the trachea and carina often provides information about extent of involvement, configuration, and axis of the airway and patency of the distal airway. This information can be invaluable when first gaining control of the airway. Obviously, patients in respiratory distress are unable to have detailed radiological assessment of their airways; a plain chest roentgenogram often will suffice.

ANESTHESIA. The anesthetic management of patients with a compromised airway is crucial to a successful outcome. In virtually every case, we have used the same technique used for patients with tracheal stenosis (the deep

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halothane inhalation technique described in detail by Wilson [1]). This technique allows the patient to maintain spontaneous ventilation throughout the procedure. Induction of anesthesia can be time-consuming and requires patience on the part of the anesthesiologist and surgeon. Any disadvantages this may impose are greatly outweighed by the tremendous advantage of having the patient spontaneously ventilating throughout the procedure, which is especially important before the exact nature of the obstruction is known. In such circumstances, the surgeon will have more time to establish an airway than if the patient were apneic. During the course of tumor removal, maintaining the airway may be difficult, and patients' ability to breathe is often the only thing that sustains them through this period. We have deliberately avoided use of muscle relaxants and agents that depress respiratory drive. Patients need to be able to clear secretions, debris, and blood from their airway postoperatively. Anesthetic agents that interfere with this function should be avoided.

ENDOSCOPIC TECHNIQUE. It is most important that the surgeon be in attendance during induction of anesthesia. Sudden compromise of the airway may occur, and immediate insertion of a rigid bronchoscope may be required to secure the airway. This demands that the surgeon and assistants be prepared and have all necessary instruments available to establish an emergency airway. We had no occasion to perform emergency tracheostomy as all patients could be intubated with a rigid bronchoscope. Knowledge of the location of the tumor makes this task much easier.

The technique of inserting a rigid bronchoscope should be familiar to all surgeons. It is important to have available rigid bronchoscopes of graded sizes (3.5 to 9 mm). A small pediatric rigid bronchoscope may be the only bronchoscope that can be inserted through very proximal, tight malignant strictures. This allows establishment of an airway; subsequently, larger bronchoscopes can be inserted. For more distal tumors, insertion of a larger bronchoscope in the proximal airway allows careful inspection and suctioning of any secretions. In most cases a No. 8 rigid bronchoscope can be passed along one side of the tumor to establish an airway and remove retained secretions. It is important to establish an airway, clear retained secretions, and stabilize the patient before proceeding with tumor removal. This allows the patient to be maximally ventilated at the start of the procedure. Inspection of the distal airway will aid the bronchoscopist in reestablishing the airway once the actual procedure has started. It is important to establish the axis of the airway when coring-out is started. The endoscopist should stay parallel to the axis at all times to avoid penetrating the wall and creating a pneumothorax or injuring the pulmonary artery.

The tumor should first be biopsied to determine its consistency and vascularity. Once this has been established, the surgeon can decide whether to attempt repeated removal of the tumor with bronchoscopic forceps or to use the tip of the bronchoscope to core out the tumor. If the tumor is believed to be of sufficient consistency and vascularity to allow a large portion of it to be removed, the rigid bronchoscope is used in a corkscrew fashion. The beveled tip of the bronchoscope is used to shave off a large piece of the tumor. Most tumors are of such consistency that shaving is easily accomplished and does not require excessive force. The anesthesiologist should discontinue assisted ventilation of the patient to avoid forcing debris and tissue more distally into the airway. The piece of tumor can then be grasped with forceps and removed or may be aspirated. Occasionally, large pieces of tumor must be held against the end of the bronchoscope with biopsy forceps and tumor and bronchoscope must be removed from the airway at the same time. The endoscopist should intermittently cease removing tumor, reestablish the airway, and allow the anesthesiologist to ventilate the patient. This will also allow the bronchoscope to tamponade any bleeding that might occur from the surface of the tumor. It is important to clean the airway of debris periodically. This may be aided by irrigation. Use of the flexible bronchoscope through the rigid bronchoscope allows suctioning at the subsegmental level.

CONTROL OF HEMORRHAGE. Major hemorrhage is uncommon and did not occur in this series of patients. Minor bleeding is easily controlled with simple measures. Irrigation with saline is the first maneuver to diminish bleeding, and frequently is all that is necessary. Dilute epinephrine (0.1 mg/mL) or epinephrine-soaked pledgets on long applicators will stop persistent oozing. Various methods of tamponade are also successful in managing any persistent bleeding that does not respond to the previous methods. Use of the rigid bronchoscope to tamponade the raw surface of a tumor is effective. For bleeding from the main bronchus or more distal airway, dental packs or Fogarty venous occlusion catheters can be used to tamponade bleeding. Fogarty venous occlusion balloons can be left in place if necessary. Use of a carefully placed endotracheal tube is also effective in controlling bleeding that persists beyond the previously mentioned measures. Placing the endotracheal tube so that the balloon is resting against the raw surface and then inflating the balloon will serve to tamponade and control any active bleeding. This measure is rarely necessary. Insulated electrodes are available to coagulate bleeding points if needed. Any coagulation abnormalities should be corrected before the procedure is performed. Use of fresh-frozen plasma and platelets at times may be necessary to correct these abnormalities.

Great care must be used to clear the airway if the patient is intubated at the completion of the procedure. The endotracheal tube may be plugged easily by blood clot. It is preferable to have the patient extubated and awake to clear the airway by coughing.

Age and Sex Distribution

There were 28 men and 28 women in this series. The median age for men was 59 years (range, 22 to 79 years), and the median age for women was 54 years (range, 20 to 81 years).

Table 1. Histological Classification of Tumors Treated

Histology	Trachea	Carina	Bronchus
Squamous carcinoma	8	11	6
Adenoidcystic	2	7	0
Adenocarcinoma	0	3	4
Thyroid	3	0	0
Sarcoma	1	1	2
Carcinoid	1	0	1
Mucoepidermoid	1	0	0
Lymphoma	0	1	0
Small cell carcinoma	0	1	1
Melanoma metastatic	0	0	1
Metastatic acinar carcinoma	0	0	1

Symptoms

All patients were symptomatic when first seen. The majority of patients experienced symptoms of shortness of breath or dyspnea on exertion (88%). The next most common symptom was hemoptysis (45%). Obstructive pneumonia was present in 18 patients. Nineteen patients had acute respiratory distress or stridor. Five patients had been treated for long periods of time for adult onset "asthma" and were on heavy doses of steroids when the diagnosis of obstruction was made. These patients have gradual onset of shortness of breath and wheezing with a "clear" chest roentgenogram. Tracheal tumors are sufficiently rare that little attention is paid to the tracheal air column, or the tumor is subtle enough and cannot be seen. Steroid administration is a contraindication to tracheal or carinal resection and must be tapered before resection.

Location

The trachea was the site of primary tumor involvement in 16 patients. Twenty-four patients had tumor involving the carina. The right main bronchus was involved in 6 patients, and the left main bronchus was involved in 2 patients. The distal airways were involved in 8 patients at the lobar or segmental level.

Histology

Squamous cell carcinoma was the most common histology in the trachea, carina, and bronchus (Table 1). Adenoid cystic carcinoma was the next most common histological finding in the carina and the trachea. The miscellaneous categories included thyroid cancers, carcinoids, mucoepidermoid tumors, sarcomas, lymphomas, and various metastatic tumors.

Initial Treatment

The majority of patients were evaluated and underwent bronchoscopy electively (61.8%). Core-out was performed at the time of resection to provide an adequate airway in some patients. The remainder of the patients had to be treated emergently to establish an airway or control symptoms (23.2%) or urgently because of postobstructive pneumonia (16%).

Results

Fifty-one of the 56 patients had an improved airway after endoscopic removal of tumor. The improvement was corroborated by subjective improvement in symptoms or radiographic appearance. Five patients had no improvement, all of whom had tumor at the lobar or segmental level. Only 2 patients required repeat bronchoscopy within 2 months of the initial successful core-out. Both patients underwent repeat bronchoscopy at seven days because of subsequent airway compromise. Additional tumor was removed in both, with substantial improvement.

There were no intraoperative deaths in this series. Four patients died within 2 weeks of endoscopy of causes unrelated to the procedure. All 4 patients had received no benefit from previous radiation therapy and were in desperate condition with postobstructive pneumonias. All patients died of respiratory failure or sepsis and could not be weaned from the ventilator.

Length of Procedure

The exact duration of the procedure was difficult to determine from review of records, but information regarding the duration of anesthesia, which did not correlate with duration of procedure, is available. Taking this factor into account, however, median length of anesthesia was 60 minutes (range, 20 to 150 minutes).

Further Therapy

Sixteen patients (28.6%) underwent resection of the tumor. There were nine tracheal resections, three carinal resections, and four pulmonary resections, including one sleeve lobectomy. The indications for endoscopic resection of the tumor varied. Some patients were originally seen with postobstructive pneumonia and had relief of the obstruction and subsequent clearing of pneumonia before definitive resection. A few patients were first examined while on high-dose steroids for presumed "asthma," thus precluding definitive surgical resection at that time. Coring out the tumor allowed the patients to return home and be weaned from the steroids; resection was performed later. A few patients had acute respiratory distress and were initially treated by endoscopy for relief of airway obstruction. Subsequent workup and successful resection were then performed. The remainder of patients had endoscopic removal of the tumor at the time of operation to allow safe management of the airway.

Most patients had unresectable disease. Thirty-four patients had other forms of treatment (60.7%). Radiation therapy was the most common additional treatment; a few patients were treated with chemotherapy or a combination of chemotherapy and radiation therapy.

Six patients had no further therapy. Four of these patients had failure of previous radiation therapy or chemotherapy and subsequently died. Two patients refused additional therapy.

Complications

Nineteen major complications were recorded in 11 patients. Five patients developed pneumonia in previously unaffected lungs after relief of postobstructive pneumonia. All of these patients responded to antibiotics and chest physiotherapy. Virtually all patients had minor amounts of bleeding. Three patients had more than minor bleeding. None lost more than 500 mL of blood, and the bleeding was easily controlled with the methods previously mentioned. Two patients developed a pneumothorax postoperatively. Only 1 of them required a chest tube. Hypoxia and hypercarbia were poorly documented in patients before the advent of CO_2 monitoring and oximetry. Two patients had documented hypercarbia and hypoxia during the course of the operation that persisted and required intubation at the end of the procedure. These 2 patients were extubated within 24 hours. Hypoxia and hypercarbia were related to blood, retained secretions, and debris. Minor arrhythmias were noted during the course of the operation in many patients. Only 6 patients required intervention beyond routine measures to control their arrhythmias. Four patients with ventricular premature beats required intravenous Xylocaine (lidocaine) for 24 hours. Atrial arrhythmias requiring digoxin were documented in 2 patients. One patient developed postoperative laryngeal edema. This responded to racemic epinephrine and steroids. Tracheostomy was avoided in this patient.

Survival

Short-term survival is directly dependent on the success of the endoscopic core-out of tumor. Long-term survival depends on the effectiveness of proposed treatments. For patients who have an obstructing lesion that requires immediate removal, coring out will allow future resection when appropriate. For inoperable malignancies, coring out will establish an airway until appropriate therapy is started. For patients who have failed previous therapies, endoscopic removal of tumor is a temporary measure that allows a patent airway. Use of tracheotomy tubes and T tubes may ultimately be effective in maintaining an airway in this group of patients who have no other options. Because of the diverse patient population reported in this series, no conclusions can be drawn regarding survival. Survival of patients who underwent resection has ranged from 5 months to more than 8 years with no evidence of disease. Survival of inoperable patients after irradiation or chemotherapy, or both, has ranged from 2 months to 82 months with a median of 6 months. Success of treatment depends on histology, location, and extent of disease.

Comment

Use of the rigid bronchoscope and biopsy forceps has been recognized as an effective method of removing endobronchial tumor since the earliest days of endoscopy. In 1915, Jackson [2] removed a fibroma from the lower margin of the left upper lobe bronchus. He concluded that "the removal of benign neoplasms of the bronchi can readily be effected by bronchoscopy." For many years, patients with unresectable neoplasms, elderly patients, or poor-risk patients were palliated reliably with this technique.

Concern about hemorrhage as a result of this technique has been expressed, but such hemorrhage has been poorly documented. Houston and colleagues in 1969 [3] and Nakratzas and co-workers in 1974 [4] reported their results with endoscopic removal of obstructing tumors of the airway. The patients in neither series experienced profuse bleeding, but these investigators perpetuated the misconception of the possibility of hemorrhage by citing earlier reports in which this complication was mentioned. No other reports in the literature have countered this unfavorable opinion. When notoriously vascular tumors such as hemangiomas, arteriovenous malformations, and some carcinoids are avoided, massive bleeding from endoscopic removal of tumors is exceedingly unusual, as in our series. Even some carcinoids can be managed in this fashion, as evidenced by the 2 patients successfully treated in our series. Adherence to the principles of the technique and an awareness of the methods to control hemorrhage should minimize the threat of hemorrhage.

Although few studies report endoscopic removal of tumor, many studies describe large series of patients treated with lasers [5–17]. Initially, most reports were from pulmonologists using yttrium-aluminum-garnet lasers through flexible bronchoscopes. Pulmonologists are now "rediscovering" the rigid bronchoscope as the preferred way to use the laser at a time when many thoracic surgeons are abandoning rigid bronchoscopy in favor of flexible bronchoscopy.

There is little doubt that the laser is an alternative to biopsy forceps and the rigid bronchoscope. Both achieve airway patency rates of approximately 90%. Critical comparison of the instruments, however, shows that the laser has few if any advantages and, in fact, may have disadvantages for the patient.

The laser was touted as a major advance because of its ability to control hemorrhage. Hemorrhage, as we have shown, is quite infrequent and easily controlled by much simpler and less expensive methods. Lasers may even be responsible for major hemorrhage, as evidenced by the 0.5% to 10% incidence of moderate hemorrhage and 1% to 3% incidence of fatal hemorrhage in most series [7–9, 12, 13, 15, 18].

Our main objections to the use of the laser are the expense (equipment, training personnel, physician, and operating room costs), length of procedure, need for repeated procedures to open airways, inferior anesthetic techniques, and unique risks (fires, hemorrhage, potential eye injuries). We consider all of these to be important factors in choosing our technique rather than the laser. It is ironic that many laser enthusiasts now report their technique of using the laser "to coagulate the mass and mechanically scoop out the tumor with the bronchoscope. The airway wall is photocoagulated for hemostasis" [8, 9]. This statement tends to support our view that the laser is unnecessary, because bleeding is rarely a problem with our technique.

Complicated airway obstructions are indeed a challenge to physicians. Complications are inherent in dealing with these problems. Strict attention to details of technique, however, should minimize complications. Our anesthetic technique of using halothane to maintain spontaneous ventilation eliminates the risks to patients posed by barbiturates, narcotics, and muscle relaxants needed for laser therapy [18–22].

The final concern about lasers is the potential for inappropriate treatment. A wide spectrum of physicians is now involved in treating airway neoplasms with lasers. Laser therapy for tracheobronchial malignancy is practiced by thoracic surgeons, otolaryngologists, and pulmonary medicine specialists. All who are involved in the care of such patients must be aware of the surgical options available to patients with malignant disease of the airway. Tracheal resections, carinal resections, and sleeve resections of the tracheobronchial tree are accepted methods of treatment and can be curative. In most situations, laser therapy is palliative only and offers no chance of cure. Patients with potentially curable lesions should not be denied the chance for cure because physicians are unfamiliar with these techniques. Input from knowledgeable thoracic surgeons should be mandatory to avoid this situation. Unnecessary delay caused by repeated laser treatments should be avoided as the opportunity for cure may be lost. Another misconception exists regarding the ability of laser therapy to treat benign tumors of the airway. Rarely does a tumor involve only the mucosa of the airway. Penetration deeper than the mucosa poses the risk of perforation of the airway wall by the laser. Resectional therapy for benign tumors is usually curative.

Use of the laser to treat obstructing tumors of the airway is indeed an alternative to the technique we have described, but such use of the laser must be justified as we believe it to be inferior in terms of cost, time, success in achieving a patent airway initially, and risk of complications to the patient and physician. Even the most ardent supporters of the laser now recognize the ability of the rigid bronchoscope to "scoop out" tumor after treating it with the laser. We believe that the laser represents an unnecessary step. Our data support greater consideration of the technique we describe.

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DISCUSSION

DR RICHARD B. McELVEIN (Birmingham, AL): Dr Mathisen describes the anesthetic technique to include deep halothane anesthesia with spontaneous ventilation; however, he also describes having the anesthesiologist stop ventilation while tissue is removed. Dr Mathisen, can you clarify whether these patients are being ventilated or assisted in their ventilation? Also, we have found the peripheral pulse oximeter to be an excellent monitoring instrument; do you find it useful?

Flow volume loops are a very sensitive indicator and an inexpensive, reproducible method of determining whether a patient has serious recurrent airway obstruction. Dr Mathisen, have you had any experience in using these?

We have patients whom we retreat, not because of failure of the first treatment, but because of regrowth of the tumor. Dr Mathisen, how many patients have you retreated by your technique? We found that if we have established the extent of the disease and retreatment is necessary, many of these patients can be treated on an outpatient basis.

In summary, Dr Mathisen has described one method of managing malignant airway obstruction. There are others. The important message is that people with airway obstruction should not be left to suffocate. Expedient care can relieve them of their obstruction and in some cases provide long-term survival. I have used the technique described by Dr Mathisen and have also had extensive experience using the laser; I find the laser technique preferable.

DR DAVID J. COHEN (San Antonio, TX): I enjoyed Dr Mathisen's presentation, but I think, in a sense, that the argument he has set up is a straw man. I believe that the real issue is whether to use laser therapy through a rigid bronchoscope or through a flexible bronchoscope. Use of the rigid bronchoscope, with either the yttrium-aluminum-garnet or the CO_2 laser, allows one to use the coring-out technique that Dr Mathisen has so eloquently described, as well as to biopsy and to maintain the airway, but it also allows the advantage of using the laser for removal of tumor. The laser, with which we have experience, has the added advantage that it can be used for coagulation.

I believe that using the laser with the rigid bronchoscope is better than using it with the flexible bronchoscope. The rigid bronchoscope does allow use of the coring-out technique described by Dr Mathisen and provides a much greater opportunity to reopen the bronchus with a single procedure rather than with the multiple procedures so often necessary when the laser is used through a flexible scope.

DR PAUL A. KIRSCHNER (New York, NY): I enjoyed the paper very much. I would like to ask a question and then make a brief comment.

Dr Mathisen, have you seen metastatic tumors of the trachea treated in this way? I have had 2 such patients. The first patient had a metastasis to the midtrachea 12 years after a right upper lobectomy for a stage I adenocarcinoma in 1971. He underwent rigid bronchoscopic removal in 1983, 1987, and twice in 1988. Radiotherapy was administered after the first bronchoscopy. He is asymptomatic now at the age of 94 years. The second patient had a polypoid metastasis to the same area which appeared 4 years after a sternotomy for a follicular thyroid carcinoma removed in 1977. The metastasis was removed with the rigid bronchoscope, and the airway has remained patent to date; she is now 84 years old. She received radioactive iodine. She has developed a metastasis to the manubrium.

Dr Mathisen, have you ever had large pieces of tumor break off and fall distally into the airway on either side during the procedure? I had one such experience; a main bronchus was blocked on one side due to recurrent metastatic carcinoma. I removed a large piece which fell off the forceps into the opposite main bronchus, obstructing it. The situation was precarious until I was able to retrieve the piece of tumor and relieve what could have been a fatal asphyxis.

We continue to use the rigid scope.

DR ROBERT J. GINSBERG (Toronto, Ont, Canada): I support use of the rigid bronchoscope and the coring-out technique, which I think is applicable to most major airway endoluminal malignant obstructions. In Toronto, we have used this technique for many years. Approximately 6 years ago, we began using the laser as the primary tool and were initially quite enthusiastic. However, we are once again using the coring-out technique for most cases, and use either the CO_2 or neodymium:yttrium-aluminum-garnet laser as a touch-up at the end of the procedure. We have found that coring is much faster and quite safe and agree with the remarks of Drs Mathisen and Grillo about the utility of this technique.

DR KAMAL G. KHALIL (Houston, TX): I would like to know more about the 2 cases of pneumothorax that occurred in this series. What was the pathogenesis of those 2 cases with this technique?

PRESIDENT GRILLO: Before Dr Mathisen closes the discussion, I'd like to add that I have used this technique for 30 years yet I do not know the number of patients I have treated. I never thought to start recording such cases until the laser came along and a reply was needed.

DR MATHISEN: I thank all of the discussants for their comments. I also recognize Dr McElvein's contributions in the area of laser use, and wish to underscore the fact that we recognize the laser as an alternative technique. One important point is that the surgeon must remain involved in the care of these patients.

At a time when many societies and specialties are worried about the loss of patients to other disciplines, I think this is one example in which, because of the lack of interest of the thoracic surgeon, some of the other disciplines, such as pulmonary medicine and otolaryngology, are undertaking the care of these patients. Not only does this represent a loss of patients to those other disciplines, but it also represents a loss of input from thoracic surgeons. This is unfortunate because in certain situations a surgical option might be available and, because a physician who is unfamiliar with the condition may not be aware of it, a patient might be denied the opportunity for cure.

Many proponents of laser bronchoscopy are beginning to reconsider its usefulness, as Dr Ginsberg has intimated. Frequently the laser is used to photocoagulate the tumor to char it, and the tip of the rigid bronchoscope is then used to scoop it out. Next, the laser is used to touch up the wall of the tumor to prevent any oozing. Yet the laser actually may not be necessary, as bleeding is not the major problem it was once believed to be.

Dr McElvein asked about assisted ventilation. With halothane anesthesia, patients are able to ventilate spontaneously at all times. At times during the course of this procedure assisted ventilation is certainly necessary, and the only reason for the anesthesiologist to discontinue assistance is to prevent pushing debris or blood further into the distal airway at the time of the coring-out procedure.

We agree that use of oximetry is very helpful in the management of these patients. I'm certain that before oximetry was used, many patients became hypoxic and we were unaware of it, yet we continued to use the procedure, not really knowing the status of patients' oxygenation. Clearly, the oximeter is of great benefit.

We do not have extensive experience using flow volume loops in patients with airway obstruction. I think it certainly is a very effective method of following them, although I think that most patients tell you when they become symptomatic.

In terms of the number of retreated patients, 2 patients were retreated at seven days because of persistent or recurrent symptoms and were found to have a tumor that could then be removed. Four other patients were retreated and represent the difference between the total number of patients and the total number of procedures. These treatments occurred at intervals ranging between 2 and 4 months. I believe that the patient who had the most procedures lived for 6 to 7 years with an adenoid-cystic tumor and required repeated endoscopic removal of her tumor at about 4-month intervals.

We have used the technique to treat exuberant granulations. Using the tip of the scope or the rigid biopsy forceps removes the granulation tissue as effectively as the laser does. I think this underscores the fact that these two methods are alternative techniques for treating the same kind of problem.

Dr Cohen raised the issue of the flexible bronchoscope and the rigid bronchoscope. If you use the laser, you should use the rigid bronchoscope, thus avoiding the risk of damaging the flexible bronchoscope by fire or melting the tip of the scope, which obviously can be very expensive. Use of the rigid bronchoscope allows the technique that we have described. If you are comfortable using the laser, I encourage you to use the technique that Dr Cohen described, including use of the rigid bronchoscope as a supplement.

Dr Kirschner asked about treatment of patients with metastatic lesions. In this series 8 or 9 patients had metastatic tumors. However, in no patient in this series did we penetrate the airway while removing the tumor.

The pathogenesis of pneumothorax in 2 patients may have been related either to intraoperative trauma or to overassisted ventilation and resultant further obstruction of the patient's airway. If one does not observe the axis of the airway and stay parallel to it, the wall of the tumor is easily penetrated and a pneumothorax is created. If the anesthesiologist is overvigorous in assisting ventilation, the airway becomes further obstructed by retained debris or secretions. High pressures might result, in turn causing a pneumothorax. We cannot tell from review of these patients' records which situation occurred.

Effect of Intraoperative Intervention on Neurological Outcome Based on Electroencephalographic Monitoring During Cardiopulmonary Bypass

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Neurological complications of cardiopulmonary bypass procedures are well documented. The present two-part study was undertaken to (1) determine if on-line computerized electroencephalographic changes correlated with neurological outcome and (2) compare neurological outcome with that of a second group of patients who received intraoperative interventions based on electroencephalographic data. Part 1 consisted of monitoring 50 patients. A power drop index was developed that correlated with new global neurological deficits. New global deficits occurred in 44% of the patients. In part 2, this information was used to design intervention criteria. Treatment protocols used previously accepted methods of increasing cerebral blood flow, ie, increasing pump flow, raising mean arterial pressure, and increasing CO_2 content in the ventilator blend. Global neurological deficits were reduced to 5% in a group of 41 clinically similar patients ($p < 0.001$). Cerebral perfusion pressures were similar in both groups. The single correlating factor was the power drop index as identified by computerized EEG. Our conclusion is that simple intervention guided by computerized EEG can reduce global neurological deficits in patients having cardiopulmonary bypass procedures.

(Ann Thorac Surg 1989;48:476–83)

Severe neurological complications after heart operations have become less common because of improvements in surgical technique, perioperative care, and extracorporeal circulation equipment [1, 2]. However, less obvious neurological deficits still occur regularly in operations using cardiopulmonary bypass (CPB) [1, 3–5]. Multiple etiological factors have been implicated, including equipment-related microemboli, intraoperative hypotension, cannula placement, and emboli from the surgical field [2, 6–9].

Several studies have attempted to determine the relative importance of these various etiological factors. These studies delineated problems in equipment and technique by manipulating one or more of the static variables associated with the bypass equipment or surgical technique [1, 10, 11]. They did not examine intraoperative intervention based on dynamic data such as cerebral perfusion acquired during the procedure.

Intact neurons produce predictable electrical patterns. Neuronal electrical activity is a direct reflection of adequacy of cerebral perfusion under adequate anesthesia. Electroencephalography (EEG) is the only direct measurement of electrical activity and therefore of neuronal integrity. Inadequate cerebral perfusion pressure (CPP) can be a result of decreased mean arterial pressure (MAP) and is immediately reflected by suppressed EEG activity. Electroencephalography has been used successfully for intraoperative assessment [12]. Data obtained from intraoperative EEG monitoring, using either the standard EEG polygraph or computerized EEG (CEEG) devices, have correlated with neurological outcome [8, 13]. However, standard EEG has been cumbersome and not well correlated with diffuse (global) neurological deficits.

A two-part study was undertaken to monitor brain activity during hypothermic cardiopulmonary bypass procedures for the purpose of (1) determining EEG characteristics consistent with neurological outcome and (2) comparing a similar group of patients who had received intraoperative interventions based on the identified characteristics.

Part 1 (group A) involved data acquisition and analysis of on-line information in an easily interpretable format.

Part 2 (group B) involved intraoperative intervention during sustained power drops as identified by the CEEG and comparison of neurological outcomes between the two groups.

Material and Methods

Over a 1-year period, 91 unselected patients undergoing heart operations were studied. Approval was granted by the hospital's human studies committee and written informed consent was obtained from participating patients. None of the patients were excluded for prior neurological symptoms or findings (40% and 41%, respectively). Part 1 of the study (group A) involved 50 patients. These same

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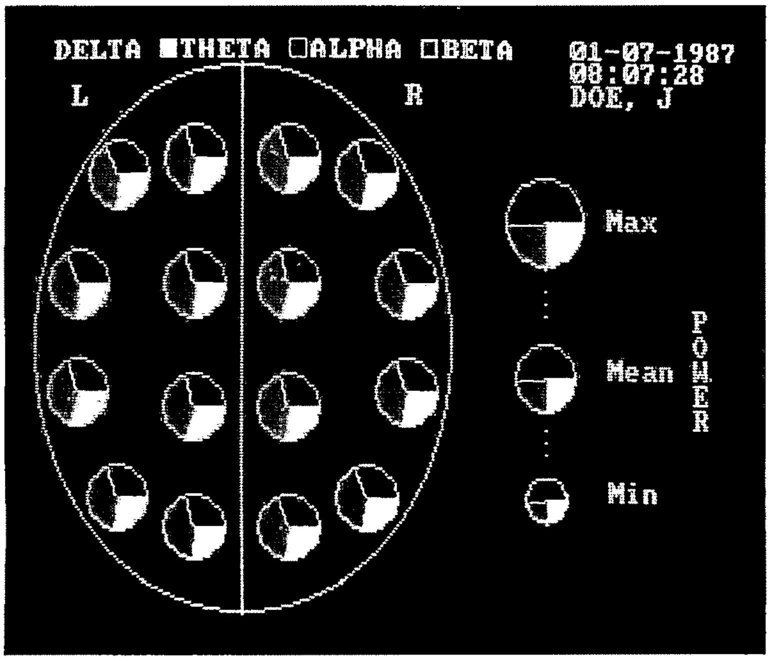

Fig 1. Pie chart display of 16 channels. (Right is right; top is anterior.) Circle size is proportional to electroencephalographic power. Colors represent the major frequency bands from slowest to fastest. Note the symmetry of size and frequency as displayed in a normal pattern.

50 patients served as the control group for part 2 of the same study.

During part 1 of the study, CEEG data were collected. Each patient was monitored intraoperatively by a 16-channel CEEG monitoring system (CNS Monitor; CNS, Inc, Minneapolis, MN). Electrodes were placed according to the international 10-20 system, and a standard bipolar anterior-posterior montage was used. These EEG data were then presented on-line in the form of pie chart and bar graphs to make interpretation easier (Figs 1, 2). The data were stored on floppy disks for review and analysis. The following intraoperative events were also recorded: MAP and central venous pressure every 15 minutes, pump flow pressures, and blood gas values.

Data analysis included (1) CEEG frequency and total power, (2) intraoperative MAP and central venous pressure, (3) arterial blood gases, (4) pump time, (5) patient's age and history of hypertension or neurological findings, or both, and (6) type of operation, ie, coronary artery bypass graft, valve replacement, or a combined procedure. Comparison of on-line data with neurological outcome was based on three perioperative structured neurological examinations.

Electroencephalographic data analysis consisted of both

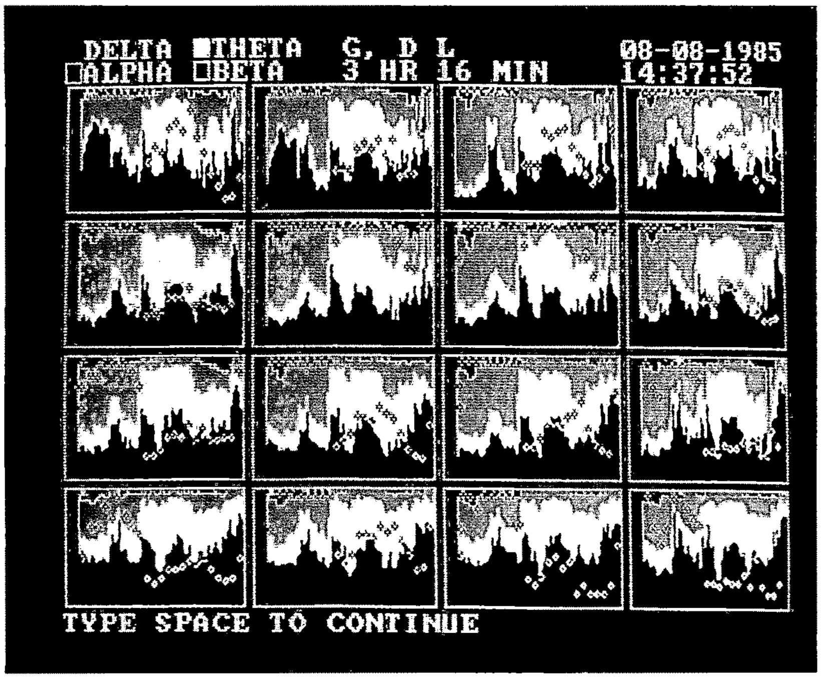

Fig 2. Frequency data; frequency bands are arranged from slowest to fastest activity: δ (red) is on the bottom, followed by θ (yellow), α (green), and β (blue) on the top. Markers (+) show relative power for each channel.

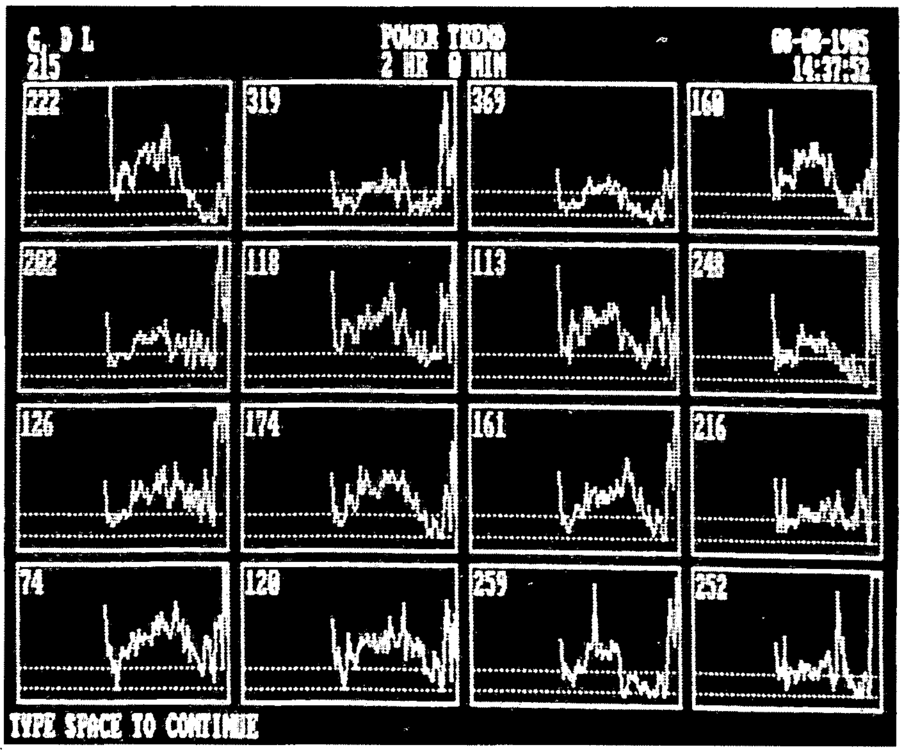

Fig 3. Illustrates 16 channels covering the whole head. (Right is right; top is anterior.) Each box represents 100% (top) to 0% (bottom) of the patients own baseline. Dotted lines *correspond with 25% and 10% power.*

frequency and total power determinations. An average total power value was calculated in each channel and overall channels (baseline) by averaging the total power from each pie chart update (20 seconds of data) from after induction of anesthesia to the onset of CPB. Total power values in each channel and an average value over all channels (averaged over one-minute intervals) from the onset of CPB to the end of operation were compared with the baseline and a power drop index (PDI) was calculated for intervals with a drop in power of at least 60% using the following formula:

$$\mathrm{PDI_n} = \mathrm{PDI_c} + \text{integer } [(40-100 \times \mathrm{P_c}/\mathrm{P_b})/5],$$

where P_c = total EEG power in the current sample, P_b = total EEG power in baseline sample (both are calculated for each channel), PDI_n = new PDI, and PDI_c = current PDI. The results are displayed in Figure 3.

Data were collected and analyzed in 2-second epochs. The EEG bands were color-coded and displayed in a pie chart and bar graph format at 20- and 60-second intervals, respectively (Figs 1, 2).

A banded average frequency value (AFV) was estimated for each channel from the band spectral data stored on diskette using the following formula:

$$\mathrm{AFV} = [\mathrm{P}(\delta) \times 2\ \mathrm{Hz}] + [\mathrm{P}(\theta) \times 6\ \mathrm{Hz}] + [\mathrm{P}(\alpha) \times 10\ \mathrm{Hz}]\ /\ \mathrm{P(Total)},$$

where P( ) = power in frequency range. An average value was calculated across all channels, from onset of the procedure to CPB onset and thereafter, yielding two measurements per patient. A ratio was calculated per patient to detect any frequency shifts. The significance of observed differences and associations was analyzed statistically using the two-sample Student's *t* test, χ^2 test, and Pearson correlation coefficient.

Anesthesia was maintained by a combination of fluorane, fentanyl, and valium unless contraindicated. Hypothermia was maintained in all but 1 patient, with body temperature lowered to 28° to 30°C as indicated by esophageal and rectal temperature probes. Preintervention MAP of 50 to 60 mm Hg was maintained in all patients according to our standards at the time of the study. Extracorporeal circulation was maintained by roller pumps with a membrane oxygenator. A 40-μm filter was used on the cardiotomy suction line. Details of the extracorporeal circuit are described elsewhere [14].

Neurological examinations were performed by an independent neurologist blind to all aspects of the study. A single neurologist performed preoperative and postoperative evaluations, thus eliminating any skewing of data because of differences in examiners. The examiner was also unaware of any differences between the two groups. The first examination was on the day before operation. Postoperative examinations occurred on day 1 (examination 1) and on day 4, 5, or 6 (examination 2). They included a standard mental status evaluation of the level of consciousness, orientation, attentiveness, memory, calculations, and speech, plus cranial nerve, motor, sensory, coordination, and reflex testing. Station and gait were included whenever possible.

Neurological outcome was classified into three categories: (1) normal, (2) global deficit, and (3) focal deficit. Patients were judged to be normal if there were no neurological changes from the preoperative assessment. Focal deficits are defined as being localized to one area of the brain. Global deficits are defined as bilateral brain

Table 1. Diagnosis of Group A Patients at Examinations 1 and 2

Diagnosis	Examination 1	Examination 2
Normal	18 (36)	32 (64)
Global	22 (44)	10 (20)
Focal	10 (20)	8 (16)

Numbers in parentheses are percentages.

Table 3. Duration of Cardiopulmonary Bypass in Group A

Diagnosis	Duration of Cardiopulmonary Bypass (min)
Normal (n = 18)	87.7 ± 16.1[a]
Global deficit (n = 22)	106.5 ± 16.7[a]
Focal (n = 10)	91.4 ± 22.0

[a] $p < 0.05$.

abnormalities exhibiting as mental status, sensorimotor, coordination, or reflex changes. Patients with a focal deficit in addition to a global deficit were assigned to the global category.

Results of Part 1

Of the 50 patients studied, there were no deaths intraoperatively or within the first week postoperatively. The number of group A patients with each diagnosis at examinations 1 and 2 is shown in Table 1.

There was a statistically significant difference ($p < 0.05$) between the diagnostic groups, normal vs global deficits, and their respective drop in global EEG power averaged over the entire head (Table 2). As the global PDI was not intended to correlate with focal abnormalities, this difference was not found to be significant when the normal group was compared with the focal deficit group. Focal abnormalities are readily visible on displays, but their study and interventions were not the focus and are beyond the scope of this article. The time on bypass was found to be statistically significant ($p < 0.05$) when comparison was made in the normal group versus global deficit group (Table 3). There was also a significant difference with relation to cerebral perfusion pressure and outcome ($p < 0.01$) (Table 4).

No significant difference was found in the normal versus focal deficit group with relation to pump time. No statistically significant difference was found between the diagnostic groupings and a history of hypertension or prior neurological symptoms. There was also no difference in outcome observed between the different surgical procedures employed.

In summary, the drop in EEG power was believed to be a significant EEG correlate of global neurological dysfunction postoperatively. Many studies have documented neurological complications that result from open heart procedures and many have changed those procedures to lower the number of complications, but none have intervened during procedures in an attempt to lower the global complications. This was our intent in the second part of our study of CPB procedures.

Purpose of Part 2

Part 2 consisted of an additional 41 cardiac surgical patients and used methods and materials identical to those described in part 1. The purpose was to determine if neurological outcome could be improved by intraoperative interventions based on CEEG information. Based on part 1 data of correlation of PDI with neurological outcome, the following criteria for and methods of intraoperative intervention were established.
Criteria for intervention:

1. Any drop in power to 25% of baseline activity during the pump run.
2. Any asymmetry or lateralized drop in power during the pump run.

Methods of intervention:

1. Increase cerebral perfusion pressure (MAP minus central venous pressure) to 60 to 65 mm Hg
    a. Increase CPB pump flow
    b. Increase MAP using Neo-Synephrine (Winthrop-Green Laboratories, New York, NY)
2. Readjust the venous cannula
3. Increase blood CO_2
4. Readjust the arterial cannula for a lateralized deficit

Interventions are based on using incremental amounts, thus avoiding overtreatment on a initial power drop. By

Table 2. Power Drop Index and Average Frequency Value for Group A

Diagnosis	Power Drop Index (mean ± standard deviation) Exam 1	Power Drop Index (mean ± standard deviation) Exam 2	Average Frequency Value Estimate (Hz) Exam 1	Average Frequency Value Estimate (Hz) Exam 2
Normal	107 ± 86 (18)[a]	154 ± 110 (32)	1.07 (18)	1.12 (32)
Global deficit	186 ± 86 (22)[a]	153 ± 47 (10)	1.16 (22)	1.15 (10)
Focal deficit	115 ± 96 (10)	89 ± 45 (8)	1.13 (10)	1.11 (8)

Numbers in parentheses are number of patients. [a] $p < 0.05$.

Table 4. Cerebral Perfusion Pressures in Group A

	Cerebral Perfusion Pressures (mm Hg)	
Diagnosis	Exam 1	Exam 2
Normal	64.4 ± 5.1 (18)[a]	63.3 ± 6.5 (26)
Global deficit	59.0 ± 6.6 (22)[a]	57.8 ± 4.6 (7)
Focal deficit	62.2 ± 7.7 (10)	61.5 ± 7.7 (18)

Values are given as mean ± standard deviation; numbers in parentheses are number of patients.

[a] $p < 0.01$.

Table 6. Comparison of Groups A and B

Variable	Group A	Group B
No. of patients	50	41
PDI	143 ± 99	71 ± 92[a]
CPB duration (min)	97 ± 25	116 ± 38[b]
Age (yr)	61.8 ± 10.5	61.8 ± 11.9
Positive preop neurological findings	40%	41%
CPP (mm Hg)	61.6 ± 5.9	59.8 ± 6.1
Length of stay (days)	12.8 ± 11.1	10.1 ± 2.4

Where applicable, data are shown as the mean ± the standard deviation.

[a] $p < 0.01$; [b] $p < 0.05$.

CPB = cardiopulmonary bypass; CPP = cerebral perfusion pressure; PDI = power drop index.

using small increments, we can achieve the desired effect at the minimal dosage for an individual patient. Single or multiple incremental interventions were used until maximal CEEG power was obtained. Aside from problems in the first five minutes of pump run, most other intervention situations call for an increase in flow rate and MAP. When intervention criteria were met, pressure or flow was maintained at or above the corrective level for a period of 30 minutes to ensure that reflow was well established.

Results of Part 2

Comparisons were made between control group A and intervention group B patients. No patients died during the course of the study. Neurological outcome was found to be considerably improved in group B when compared with group A. The difference resulted from a decrease in the number of global complications (Table 5). The effect was most significant ($p < 0.001$) for the first postoperative examinations. There were no significant differences between diagnostic groupings and CPP (Table 6).

A number of significant statistical differences were found between groups A and B (Table 6). The mean PDI for group A was significantly higher ($p < 0.01$) than that of group B. The mean CPB duration also differed significantly ($p < 0.05$). A significant ($r = 0.408$, $n = 50$, $p < 0.01$) positive correlation was found between the averaged PDI and CPB duration in group A. In group B, no significant ($r = -0.107$, $n = 41$, $p < 0.1$) correlation was observed. Although there were longer pump runs in group B, the average PDI was lower. The mean age and history of prior neurological symptoms were quite similar in the two groups. The average CPP during CPB in each group was almost identical (Table 6). Postoperative length of stay was 12.8 ± 11.1 days for group A and 10.1 ± 2.4 days for group B.

The types of surgical procedures between groups were comparable although there was a slightly higher percentage of valve procedures performed in group B patients (Table 7).

Table 5. Results in Both Groups

	Exam 1		Exam 2	
Diagnosis	Group A	Group B	Group A	Group B
Normal	36% (18)	76% (31)	64% (32)	80% (33)
Global deficit	44% (22)[a]	5% (2)[a]	20% (10)	2% (1)
Focal deficit	20% (10)	19% (8)	16% (8)	17% (7)[b]

Numbers in parentheses are number of patients.

[a] $p < 0.001$. [b] Two patients experienced postoperative strokes that resulted in focal deficits.

Case Reports

Group A, Patient 37

This patient is a 51-year-old right-handed man with no prior neurological complaints. A preoperative neurological examination revealed a slight hyperreflexia of the right lower extremity without other abnormalities. The patient underwent a three-vessel coronary artery bypass graft.

Postoperatively on the first examination, the patient exhibited slowed mentation with mild memory dysfunction and reduced performance on calculation testing. Deep tendon reflexes were diffusely brisk. No focal findings were noted. On the second examination, the patient continued to evidence signs of a mild encephalopathy without a focal deficit.

The power trend reveals a diffuse drop in CEEG power with an averaged PDI of 243. The increase in power noted midway in the graph represents the increase in CEEG power associated with rewarming (Figure 4).

Group B, Patient 40

This patient is a 72-year-old right-handed man who had no prior neurological symptoms and who had a normal

Table 7. Surgical Procedures

Procedure	Group A	Group B
Coronary artery bypass grafting	76%	63%
Valve replacement	18%	27%
Both	6%	10%

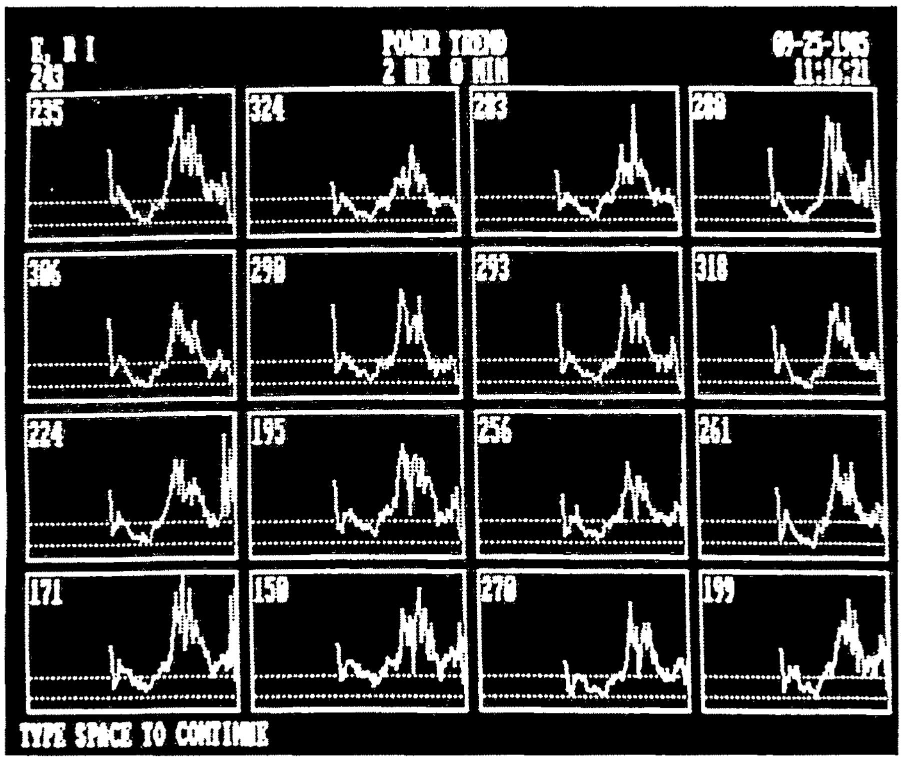

Fig 4. This power trend demonstrates a diffuse drop in power as compared with the baseline. This patient suffered from a diffuse encephalopathy postoperatively.

preoperative neurological examination. The patient underwent an aortic valve replacement and a three-vessel coronary artery bypass graft. Both first and second examinations were normal.

The power trend reveals an initial large drop in EEG power diffusely. This coincided with a drop in cerebral perfusion pressure to 33 mm Hg. The MAP was elevated into the range of 50 to 60 mm Hg with only minimal changes in CEEG output. Therefore, the MAP was increased further to 80 to 90 mm Hg, with considerable improvement in CEEG output (large increase in the middle of each graph). The average PDI by the end of operation was 72 (Figure 5).

Group B, Patient 38

This patient is a 67-year-old right-handed woman with no prior neurological deficit. She underwent a four-vessel coronary artery bypass graft with no postoperative neurological deficits.

The power trend shows a prolonged but mild power drop. The PDI by the end of operation was only 30, reflecting the relatively mild drop. In this case, the average CPP during CPB was only 43 mm Hg. Despite the low CPP, intervention was not necessary as the EEG remained acceptable (Figure 6).

Comment

Electroencephalographic monitoring during CPB has been advocated since the early 1970s [12, 13, 15]. Although this technique will not detect all clinically significant lesions affecting the cerebrum and brainstem, it has been shown to be a sensitive indicator of neuronal function [16–18].

Using eight-channel EEG recorders Salerno and associates [8] detected diffuse EEG changes related to low perfusion pressures and superior vena cava obstruction from an unsuspected kink in the venous lines. Multichannel recordings also detected a focal area of dysfunction that resolved after repositioning the aortic arch cannula.

Using eight- and nine-channel electroencephalographs, Wright and co-workers [13] described patterns of EEG activity believed to be associated with the onset of CPB, venous hypertension, reduced perfusion pressure, embolism, and hypoxia. Unfortunately, these EEG conventional recordings required interpretation by an electroencephalographer. As these patterns are not characterized in a quantitative fashion, it becomes difficult to construct protocols whereby rapid intraoperative interventions can be employed.

Applying digital computer techniques to the analysis of EEG has increased the sensitivity for the detection of EEG changes related to ischemia. Stockard and associates [12], using six to 14 channels of CEEG data analysis, also found a correlation between intraoperative hypotension, postoperative neurological complications, and EEG dysfunction. They demonstrated an age dependence of the cerebral vulnerability to hypotension and attributed this to either increasing carotid and intracranial atherosclerosis or diminished neuronal reserve. Neurological complications were found to correlate with the duration of CPB.

Aberg and colleagues [19], using adenylate kinase measurements in the cerebral spinal fluid, indicated that 50% to 60% of patients undergoing CPB have evidence of ischemic brain lesions. Their results are comparable with the present findings in group A, examination 1, and

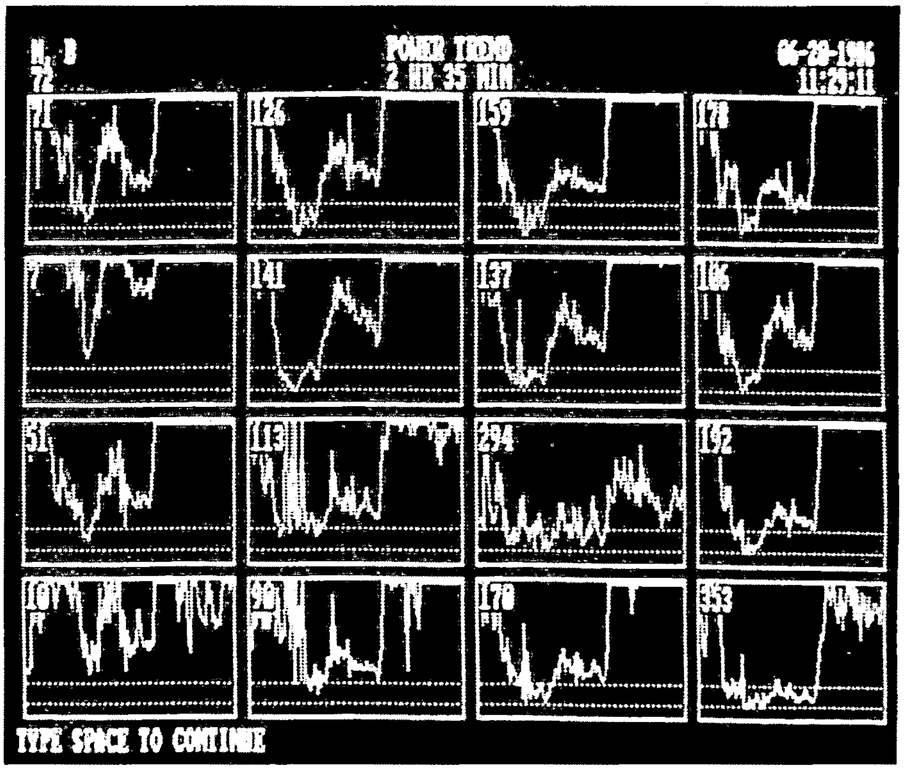

Fig 5. This power trend demonstrates an initial power drop associated with a decrease in cerebral perfusion pressure at the onset of cardiopulmonary bypass. The improvement that followed corresponded to an increase in cerebral perfusion pressure. The patient was neurologically normal postoperatively.

consistent with other studies using biochemical indicators of neurological damage (20).

In this study, it was found that despite the significantly longer CPB durations in group B, the complication rate was reduced, indicating maintenance of adequate cerebral perfusion. The age dependence of cerebral vulnerability coupled with the finding of almost identical CPP in groups A and B seems to indicate no single cerebral perfusion pressure will satisfy all patients. Consequently, individualized treatment based on measurements of organ perfusion would prove superior.

Our data document the correlation of CEEG power

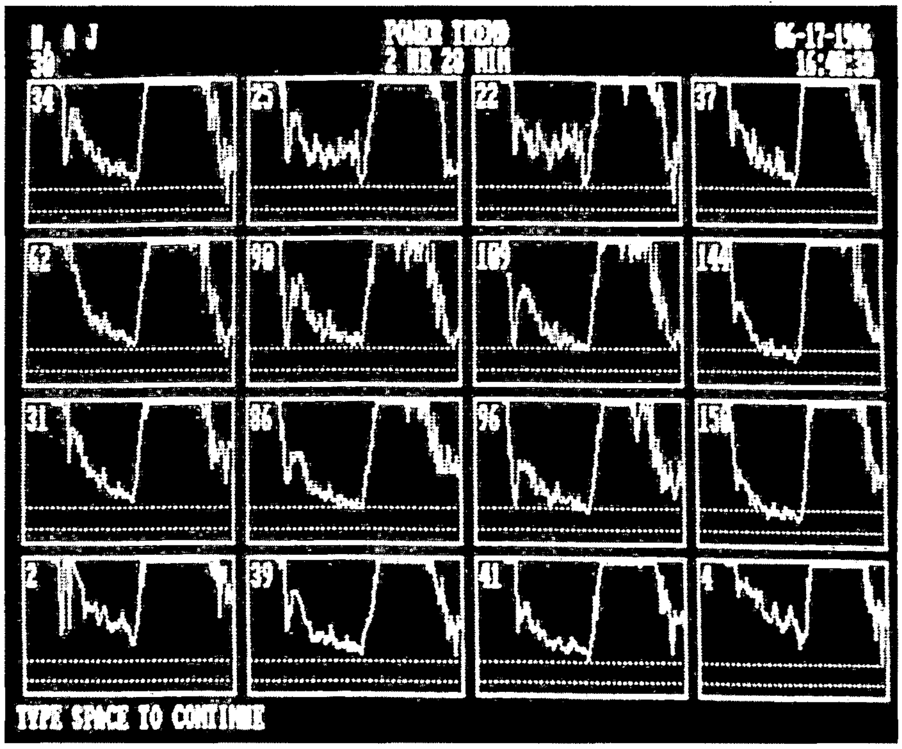

Fig 6. This patient maintained adequate electroencephalographic activity during cardiopulmonary bypass despite a relatively low cerebral perfusion pressure. Therefore, no intervention was necessary.

drop with postoperative global neurological deficits. We were able to demonstrate that on-line recording of CEEG during cardiopulmonary bypass procedures produces characteristic patterns in an easy to interpret format. Global neurological dysfunction was demonstrated in 44% of the control group. This percentage is consistent with those of other studies.

Although the reduction of rate of neurological deficits in group B to 5% is quite remarkable, it is worth noting that with a sample of only 41, the 95% upper confidence bound on the true rate of neurological deficits is higher than 5%. That is, it could be entirely possible with these results to find a much higher rate in another study with a similar population. Also, there is usually increased statistical power when a continuous measure such as PDI is analyzed as a continuous variable to the neurological outcome, but in this study, this measurement was not carried out.

Length of pump time has previously coincided with an increase in neurological deficits. In this study it was found that despite longer CPB duration in group B, the complication rate was reduced, reflecting adequate cerebral perfusion. The type of procedure, age, and prior history of hypertension or neurological symptoms were not significant factors in this group.

Hypotension and low cerebral perfusion pressures have previously been associated with global deficits. These are also the factors that may be most amenable to treatment.

Patterns and levels of significant power drops can be used to establish criteria for intraoperative interventions designed to restore adequate cerebral blood flow to avert brain injury.

In our opinion, intraoperative interventions based on dynamic CEEG data offer an opportunity to reduce the percentage of global neurological deficits occurring during CPB. Further studies will continue to refine characteristics of adequate cerebral blood flow and CPP and corrective interventions.

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Improved Cardiac Function With Glucose-Insulin-Potassium After Aortocoronary Bypass Grafting

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To assess the effectiveness of metabolic support for the heart in patients with refractory heart failure after hypothermic ischemic arrest for aortocoronary bypass grafting we assigned 22 patients to receive either intravenous glucose (50%), insulin (80 IU/L), and potassium (100 mEq/L) at a rate of 1 mL/kg/h for up to 48 hours (GIK) or glucose (5%) and NaCl (0.225%) at the same rate (control). All patients started out with a mean cardiac index of less than 3.0 L/min/m^2, were on intraaortic balloon pump assistance, and required inotropic drugs. At 12 and 24 hours cardiac index had increased significantly in the GIK group when compared with the control group (3.6 and 3.4 versus 2.5 and 2.7 L/min/m^2, respectively). Time on the intraaortic balloon pump (39 versus 61 hours) and requirements for inotropic drug support were significantly less in the GIK group than in the control group. All 11 GIK patients could be weaned from intraaortic balloon pump assistance. At 30 days after operation survival was 10/11 in the GIK group, compared with 7/11 in the control group. We conclude that GIK is both safe and effective in the treatment of refractory left ventricular failure after aortocoronary bypass grafting. The exact mechanism for the beneficial effect of GIK on myocardial contractility remains to be elucidated.

(Ann Thorac Surg 1989;48:484–9)

In recent years refractory left ventricular pump failure after hypothermic ischemic arrest has continued to carry a consistently high mortality rate. The experience of the Texas Heart Institute during the years 1985 to 1987, summarized in Table 1, indicates that those patients requiring hemodynamic support by intraaortic counterpulsation are on average at a 36% risk of death in the immediate postoperative period.

We have recently observed in the laboratory that "glycogen loading" before hypothermic ischemic arrest of rabbit hearts improves their tolerance to ischemia [1]. Although several clinical reports suggest that metabolic support with glucose and insulin may improve left ventricular function in myocardial ischemia [2–5] or left ventricular failure of other etiology [6–8], there are to date no prospective studies examining the efficacy of this intervention in patients exhibiting refractory left ventricular failure after cardiopulmonary bypass and hypothermic ischemic arrest for aortocoronary bypass grafting.

In spite of the diversity of opinion and intense speculation surrounding the metabolic and physiologic derangements that persist in myocardial tissue after the resolution of an ischemic event, the concept of metabolic support has gained interest in the face of a more accurate description of derangements that characterize ischemic myocardium. In a broader sense, the term *stunned myocardium* [9] not only denotes reversible myocardial dysfunction caused by ischemia and reperfusion after an acute coronary artery occlusion, but can also be applied to transient functional abnormalities seen after hypothermic ischemic arrest. Although improved methods of cardioplegia have been the focal points in improving myocardial preservation in operations requiring an arrested heart [10], metabolic interventions in vivo have until now received no attention.

With these considerations in mind, we designed an alternate-assignment clinical trial using infusion of glucose, insulin, and potassium in patients with refractory left ventricular failure after aortocoronary bypass grafting. We thought that the postoperative administration of glucose, insulin, and potassium would improve left ventricular function, shorten time on the intraaortic balloon pump, and enhance postoperative survival.

Table 1. Postoperative Survival in Patients With Refractory Left Ventricular Failure at The Texas Heart Institute

Year	Number of Patients	No. of Patients Weaned	No. of Patients Who Died
1985	119	77 (66)	42 (34)
1986	157	102 (65)	55 (35)
1987	159	100 (63)	59 (37)
Total	435	278 (64)	156 (36)

All patients required intraaortic balloon counterpulsation support. Numbers in parentheses are percentages.

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Table 2. Demographic Data

Group	Mean Age[a] (yr)	Males	Females	Total
Control	64 ± 7	6	5	11
GIK	60 ± 10	9	2	11

[a] Data are shown as the mean ± standard deviation.

GIK = glucose-insulin-potassium.

Material and Methods

Patient Population

The study population consisted of 22 patients undergoing aortocoronary bypass grafting at the Texas Heart Institute from November 23, 1987, to April 5, 1988. During the same period altogether 47 patients were treated with intraaortic balloon support, of which 25 were excluded because of operation for valvular heart disease or because of the exclusion criteria set forth in this section. Demographic data are presented in Table 2. The study was reviewed and approved by the Human Subjects Review Committee of the Texas Heart Institute and St. Luke's Episcopal Hospital. All patients entered into the study had been on cardiopulmonary bypass for a period of up to 45 minutes and on cold ischemic cardioplegic arrest during the performance of bypass graft anastomoses. The method of cardioplegia at the Texas Heart Institute was the same in all patients and has been described by Cooley [11].

All patients were enrolled after the independent determination by the operating surgeon that mechanical hemodynamic assistance (in all instances a balloon counterpulsation device) was indicated. One of the principal investigators was then notified that a patient required an intraaortic balloon pump after aortocoronary bypass grafting. Informed consent was obtained from the patient's next of kin. Patients were assigned consecutively to receive either an intravenous infusion of glucose, insulin, and potassium (GIK) or a control solution (ie, nonrandomized, alternate assignment). Subjects assigned to the GIK treatment group received a solution of 500 g of glucose, 80 U of human insulin, and 100 mEq of potassium chloride per liter of infusate at a rate of 1 mL/kg/h. The control group received 5% dextrose in 0.225% sodium chloride solution at the same infusion rate based on body weight. The study was not blinded, because in the present setting it was not possible to conceal the high plasma glucose concentrations of the treated group.

The GIK and control infusions were begun within two to six hours after arrival to the intensive care unit. Treatment was continued until the patient was weaned from intraaortic balloon pump or until 48 hours of therapy had elapsed. Patients were excluded from the study if cardiopulmonary bypass time exceeded 45 minutes, as well as if either the patient was weaned from the intraaortic balloon pump or death occurred within the first three hours after operation. Patients with renal or hepatic failure (as defined by a serum creatinine level greater than 3 mg/dL or a blood urea nitrogen level greater than 50 mg/dL, or a serum bilirubin level greater than 3 mg/100 mL) were also excluded. Glucose-insulin-potassium and control infusions were administered concomitantly with standard inotropic agents, diuretics, and antiarrhythmic medications.

Measurements

Continuous measurements of systemic arterial blood pressure, pulmonary capillary wedge pressure, thermodilution cardiac output, systemic vascular resistance, urinary output, core body temperature, and electrocardiographic trends were recorded for the analysis of the hemodynamic effects of the treated and control groups. Laboratory measurements of blood glucose, potassium, phosphate, lactate, blood urea nitrogen, creatinine, creatine kinase, lactate dehydrogenase, osmolality, and electrolytes were obtained at 0, 6, 12, 24, and 48 hours after treatment was initiated. Insulin and free fatty acid levels were also measured for most patients after treatment was started. The calculated cardiac index, intraaortic balloon pump time, and estimated pharmacologic inotropic support were recorded and used as variables of left ventricular function. An "inotrope score" was calculated from the number and amount of pharmacologic agents used including dopamine, dobutamine, amrinone, isoproterenol, and epinephrine. Details are described in the Results section.

All data presented are the mean ± the standard deviation. A two-tailed paired *t* test was used to analyze the difference between the treated and control groups for statistical significance.

Results

Mortality

Table 3 shows data on early and late mortality. Two of 11 control patients could not be weaned from intraaortic balloon pump assistance and subsequently died within 48 hours of operation, whereas all of the 11 patients treated with GIK were successfully removed from mechanical hemodynamic support. There was one late death in the GIK-treated group and two late deaths in the controls at 30 days. Late deaths in both groups were due to either sepsis or pneumonia. At 30 days after operation there were ten survivors in the treated group compared with seven survivors in the control group. Although the small numbers preclude any statistical analysis of significance, the data suggest a trend toward increased survival in the GIK-treated group.

Table 3. Early and Late Mortality

Group	Mortality	
	Early (not weanable from IABP)	At 30 Days
Control	2/11	4/11
GIK	0/11	1/11

GIK = glucose-insulin-potassium; IABP = intraaortic balloon pump.

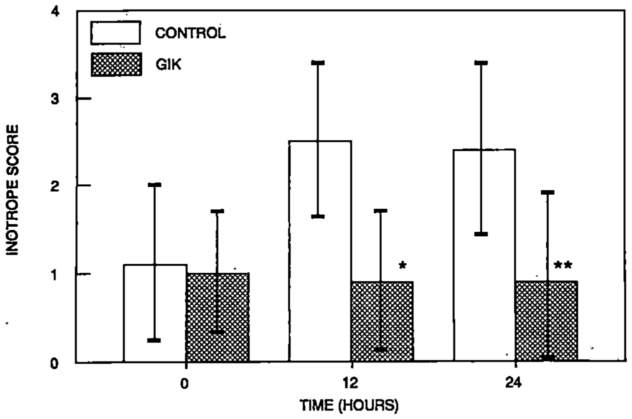

Fig 1. Mean inotrope score and standard deviation at 0 (pretreatment), 12, and 24 hours. See the Results section for the definition of inotrope score. (GIK = *glucose-insulin-potassium;* *p < *0.001;* **p < *0.005.)*

Time on Intraaortic Balloon Assist

The mean time required for intraaortic balloon assist was 39 ± 26 hours in the treated group compared with 61 ± 41 hours in the control group ($p < 0.01$).

Inotropic Support

We devised an inotrope scoring system to assess the collective requirements of each group on pharmacologic inotropic support. The scoring system assigns a value corresponding to the number of inotropic agents used in pharmacologic doses for each patient at each designated time period. If no inotropic agents were required for a particular patient at a designated time point, a value of 0 was assigned. A value of 1 was given if either dopamine (at a dose greater than 5 μg/kg/min), dobutamine, or amrinone was used alone. If both dopamine and dobutamine or a similar combination of two agents were used, that point received a value of 2. When either epinephrine or norepinephrine was used in combination with pharmacologic doses of two other agents (dopamine, dobutamine, or amrinone), a value of 3 was assigned. A value of 4 was given when four or more agents were used in combination.

As shown in Figure 1, inotrope scores were similar at 0 hours, suggesting similar requirements of the two groups in the pretreatment phase. At both 12 and 24 hours after treatment, however, the control group required significantly greater quantities of inotropic agents for hemodynamic stability (defined by a systolic blood pressure of more than 100 mm Hg) than the GIK group (inotrope scores of 2.5 ± 0.9 and 2.4 ± 1.0 versus 0.9 ± 0.8 and 0.9 ± 1.0, respectively).

Heart Rate, Systemic Vascular Resistance, and Pulmonary Capillary Wedge Pressure

When sampled at the designated time points the heart rate ranged from 98 ± 14 to 109 ± 11 beats per minute in both groups throughout the first 24 hours, and the difference between groups was statistically not significant.

Table 4. Changes in Systemic Vascular Resistance During the First 24 Hours

	Systemic Vascular Resistance[a]		
Group	0 h	12 h	24 h
Control	1276 ± 577	1075 ± 320	995 ± 303
GIK	1223 ± 703	716 ± 273[b]	739 ± 193

[a] Data are shown as the mean ± standard deviation, units are dynes · s · cm^5.
[b] $p < 0.01$.

GIK = glucose-insulin-potassium.

Mean values for systemic vascular resistance were calculated for each time point in the two groups. Each group demonstrated similar initial values of systemic vascular resistance (Table 4). The control group subsequently displayed elevated values of systemic vascular resistance when compared with the treated group, which probably reflects the greater number and amount of vasoconstricting inotropic agents required in controls (see the section on Inotropic Support).

To evaluate the possibility that the significant changes in left ventricular performance were secondary to differences in preload, the pulmonary capillary wedge pressure for each group was compared. There was no significant difference in the pulmonary capillary wedge pressure values, which ranged from 13 to 18 mm Hg (data not presented).

Cardiac Index

Cardiac indexes for the control and treated groups are presented in Figure 2. Initial mean values were similar for both groups, but subsequent values demonstrated a statistically significant increase in the treated group when compared with controls. The difference was also present at 24 hours, albeit smaller. The changes in cardiac index over time for the control and treated groups are also presented in Figure 3 as results of analysis of variance of paired data to ensure that the overall results are not

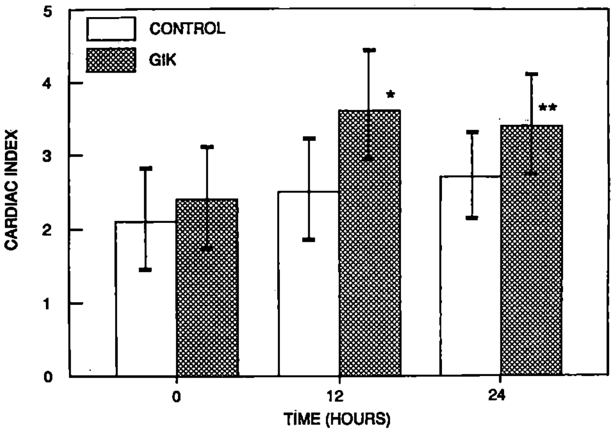

Fig 2. Mean cardiac index and standard deviation expressed in liters per minute per square meter of body surface area at 0 (pretreatment), 12, and 24 hours. (GIK = *glucose-insulin-potassium;* *p < *0.025;* **p < *0.005.)*

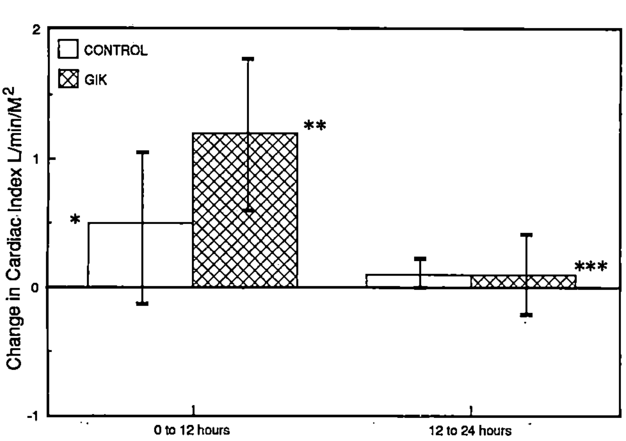

Fig 3. Mean change in cardiac index, expressed in liters per minute per square meter of body surface area. Statistical significance was determined using analysis of variance of paired samples within each group: *p = *NS;* **p < *0.001;* ***p = *NS for either group.* (GIK = *glucose-insulin-potassium.)*

derived from the performance of a few extraordinary responders.

There was a significant change in cardiac index in paired samples of the GIK-treated group in the first 12 hours of treatment. The cardiac index of the control group did not change significantly in the corresponding 12-hour period. Neither group demonstrated a significant change in cardiac index from 12 to 24 hours.

To assess the efficacy of GIK therapy in varying degrees of left ventricular failure in this setting, patients of each group were subdivided into those with an initial cardiac index of less than 2.5 L/min/m^2 and those with an initial cardiac index between 2.5 and 3.5 L/min/m^2 (Table 5). A significant difference was again found in cardiac index of the GIK-treated group when compared with the control group for each category of left ventricular failure based on initial cardiac index.

Metabolites in Arterial Blood

All patients were hyperglycemic after arrival to the intensive care unit due to glucose priming of the cardiopulmonary bypass circuit (data not presented). Glucose values remained high (greater than 250 mg/dL) in the GIK-treated group, whereas glucose concentration in controls decreased several hours after cardiopulmonary bypass was discontinued.

Table 5. Cardiac Index

Group	Cardiac Index (L/min/m^2) 0 h	12 h	24 h
Group 1 (initial CI < 2.5)			
Control (n = 8)	1.8 ± 0.4	2.4 ± 0.8	2.7 ± 0.7
GIK (n = 4)	1.8 ± 0.5	3.3 ± 0.7^a	3.1 ± 0.2^b
Group 2 (initial CI > 2.5)			
Control (n = 3)	3.0 ± 0.3	2.3 ± 0.2	2.7 ± 0.5
GIK (n = 7)	2.8 ± 0.3	3.8 ± 0.9^a	3.6 ± 0.8^c

Data are given as mean ± standard deviation. ^{a} $p < 0.005$; ^{b} $p < 0.01$; ^{c} $p < 0.05$.

CI = cardiac index; GIK = glucose-insulin-potassium.

Insulin levels were at least tenfold higher in the treated groups (in excess of the upper range of the radioimmunoassay) than in controls. It is of note that the insulin levels in controls were not remarkably elevated from the reference range in spite of substantial hyperglycemia in all patients in the immediate postoperative period (data not presented).

In both groups lactate levels were elevated compared with a reference range of 3 to 7 mg/dL at our laboratory, but there was no difference between the two groups. Free fatty acid levels were suppressed in the treated group, as might be expected from the antilipolytic effect of insulin (data not presented).

Comment

We report improvement of left ventricular function and a trend toward a reduction in mortality of patients with refractory heart failure after hypothermic ischemic arrest for aortocoronary bypass grafting. Other conditions for which metabolic support of the heart with GIK has been used include derangements in the electrical stability of the myocardium in the course of an acute myocardial infarction (the "polarizing solution" of Sodi-Pallares and associates [12]), frequent bouts of chest pain in patients with chronic angina pectoris [13], and decreased left ventricular function in acute ischemia [14]. Although it was shown that GIK decreases the extent of myocardial necrosis after coronary artery ligation in animals [15, 16] and may counteract early tissue metabolic deterioration in the infarcting baboon heart [17], two large-scale trials reported no difference in long-term survival of patients with acute myocardial infarction between GIK-treated and control groups [18, 19]. The present report has to be considered in the context of a special indication for glucose, insulin, and potassium as agents providing metabolic support for the failing heart on reperfusion after global hypothermic ischemia. In other words, although coronary artery disease was the underlying disease in all of our patients and undoubtedly contributed to the propensity for contractile failure, the acute event differed substantially from acute regional myocardial ischemia caused by coronary artery occlusion.

Conflicting data on the use of GIK in patients with acute and chronic ischemic heart disease [18, 20] have discouraged the widespread use of metabolic support for the failing heart. Concerns arising from negative clinical studies were fueled by the biochemical reasoning that glucose might be toxic to cells if reoxidation of NADH does not occur, and that under these circumstances adenosine triphosphate might be consumed for the production of fructose 1,6-diphosphate [21]. As the glycolytic pathway is inhibited further downstream, accumulation of H$^+$ ions and lactate (which, in turn, inhibit glycolysis and glycolytic stimulation by glucose and insulin) may occur. The data indicate a protective effect of glucose and insulin on the ischemic myocardium as suggested by Apstein and co-workers [22]. Our findings also lend support to Opie's hypothesis that "glucose may be good for the survival of ischemic myocardium" [23], not only by increasing the rate of anaerobic glycolysis, but also by lowering circulating free fatty acid levels, by stabilizing membrane functions and preventing loss of intracellular potassium [24], and by preserving intracellular glycogen stores [25].

Although Neely and Grotyohann [26] reported a beneficial effect of glycogen depletion in reperfused myocardium, we [27] have recently found a marked decline in left ventricular function on reperfusion of glycogen-depleted hearts after prolonged (30 to 60 minutes) normothermic or hypothermic ischemia. Indeed, there seems to be increasing evidence that glycogen has a protective role in myocardial ischemia [1, 28, 29]. Whether GIK stimulates glycogen synthesis in human myocardium, as is the case in animals [1], and, if so, whether glycogen has any function in addition to providing endogenous carbohydrate substrate during periods of oxygen deprivation remains a matter of speculation, as the metabolic consequences of varying degrees of ischemia and reperfusion on glycogen and carbohydrate metabolism have not yet been examined in detail [30].

In summary, increasing blood glucose and insulin levels appear to exert a beneficial inotropic effect on the reperfused, failing myocardium after hypothermic ischemic arrest. This effect is manifested by trends toward an increase in cardiac index, a decrease in requirements for inotropic drugs, and a decrease in 30-day mortality. The power of our conclusions is weakened by the comparatively small number of patients enrolled in the present study. A definitive study will have to include a much larger sample to satisfy statistical requirements. Although the exact mechanism for the beneficial effect of GIK on myocardial contractility also remains to be elucidated, it can be stated that GIK appears to be both safe and effective in the treatment of refractory left ventricular failure after aortocoronary bypass grafting. We hope this initial experience will provide other investigators with the basis for a multicenter, randomized, and (if technically possible) blinded trial.

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Notice From the Southern Thoracic Surgical Association

The Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association will be held at the Phoenician Golf and Tennis Resort, Scottsdale, AZ, November 9–11, 1989. There will be a $155 registration fee for the Scientific Session for nonmember physicians except for guest speakers, authors and coauthors on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 9, 1989. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Advance registration forms, hotel reservation cards, and details regarding transportation arrangements will be mailed to Association members. Nonmembers should write to the Secretary-Treasurer, Gordon F. Murray, MD.

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Effect of Crossover on the Statistical Power of Randomized Studies

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Randomized studies involving long-term follow-up are vulnerable to the effects of unplanned crossover. In surgical studies, such crossover usually occurs when control patients become more symptomatic and undergo operation. In several large studies of coronary bypass grafting, crossover ranged from 25% to 38%. The most common way of dealing with this problem is to apply the "intention-to-treat" principle, which analyzes such crossovers with their originally assigned groups. Besides the logical problem of counting a control patient who actually undergoes operation as "nonsurgical," a more subtle problem arises in terms of statistical power. When statistical power is low, a truly effective treatment may be mistakenly labeled as no better than control, causing a potentially valuable form of therapy to be ignored or discarded. This analysis demonstrates that crossover may have a profound effect on the statistical power of randomized studies and presents a method for predicting the effect of such crossover on statistical power.

(Ann Thorac Surg 1989;48:490–5)

Randomized clinical studies of surgical therapy often involve follow-up lasting many years. During the period of follow-up, patients in the control group may cross over and actually undergo operation. This crossover affects both the results of the study and their interpretation.

Crossover and the Intention-to-Treat Principle

When there is crossover in a randomized study, a problem arises regarding analysis of the results. Because patients who have undergone operation cannot cross over to the control group, crossover occurs primarily in control patients. In addition, patients who are assigned to operation but refuse and later die are counted as surgical deaths even though no operation was performed.

There are several alternatives in analyzing such patients. Our discussion takes place in the context of what must be considered the standard situation, namely, in the presence of substantial variability in the risk of death from patient to patient.

1. Drop the crossovers from the study at the time of crossover. This method alters the original balance of the two groups, thereby sacrificing the benefits of randomization. Typically, as control patients usually cross over because they have become sicker, a bias is created in favor of control by withdrawing the patients most likely to die from the control group.
2. Maintain the crossovers in their original group until the time of crossover, and then count them with the new group. Again, the original equivalence of the groups is destroyed. Bias is introduced if patients remain in the control group until they are moribund and then switch to the treatment group at a time when it is too late for treatment to be effective. The bias in favor of control is compounded further by counting these moribund patients against treatment.
3. Use the "intention-to-treat" principle. This method of analysis was popularized by Peto and colleagues [1]. The intention-to-treat principle counts crossovers with their originally assigned groups regardless of the treatment actually received, thus maintaining the original structure of the groups created by randomization. This technique permits statistical analysis of the results by standard methods. Nonetheless, a bias may still be created toward the null hypothesis in the following manner: if a "nonsurgical" patient actually undergoes operation and is saved by it, he or she will still be counted as a success of nonsurgical therapy. This creates a bias in favor of control when operation is truly better than control. (If operation were truly equal to control, crossover would have no effect on outcome; if operation were truly inferior to control, this important fact might be overlooked.)
4. Discard the study. This alternative is rarely mentioned when the intention-to-treat principle is discussed. Nonetheless, one must recognize that not all randomized studies can guarantee results that are both valid and clinically applicable.

Although the intention-to-treat principle protects the benefits of randomization, according to May and colleagues [2] ". . . randomization does not ensure the validity of the trial; it is only one of several measures that attempt to minimize systematic error and the associated benefits can easily be lost."

It has been said that the intention-to-treat principle actually tests a policy of treatment rather than treatment

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itself. This semantic distinction can readily degenerate into sophistry: a study that purports to test "treatment" versus "no treatment" must actually do so. To do otherwise is a distortion of both language and logic. This is precisely what occurs when the aim of the study is to test the efficacy of operation, but, due to crossover, the "nonsurgical" group actually consists of patients who have undergone operation. Such a study actually compares one surgical group with another surgical group. In this situation, one would hardly expect to find a striking difference in outcome. If it is the intention of the research plan to compare, say, deferred operation with immediate operation, then this should be a prespecified goal with rigorous definitions and criteria applied as part of the study protocol.

There has been much misunderstanding of the intention-to-treat principle and the importance of statistical power. Peto and colleagues [1] described trials of cancer therapy in which there was crossover by only "a few" patients and in which a great deal of attention was paid to assuring appropriate statistical power. Their warnings are too often ignored.

> The practical conclusion is that clinical trials can easily monitor death rate ratios between 2 treatments which are 1:3 or better, but that detection of anything less extreme than 2:3 is very difficult. These summary ratios are very important, and should be written on the shirt-cuffs of all trial organizers, as attempting to study a difference which could not plausibly be as extreme as 2:3, by a clinical trial, is a common mistake. . . . Clinical trials in which the influence of treatment on time to death is of prime interest should rarely be undertaken unless *either* there is some hope that the death rate can be halved, or the trial will be able to continue until at least 100 patients have died. . . [1].

Some investigators have mistakenly adopted the intention-to-treat principle as license to ignore crossover, no matter what its magnitude. This idea is insupportable. Clearly, if patients do not comply with their assigned treatment, little benefit can be anticipated and interpretation of the results is compromised [2]. If the aim of the study is to compare medical and surgical treatment, patients who cross over from medical treatment to undergo operation when their condition deteriorates violate the protocol. If such crossover is sufficiently great, the study becomes meaningless. Obviously, if crossover were 100%, no useful conclusions could be drawn.

It is difficult to quantify the point at which crossover invalidates a study, but investigators should carefully consider the frequency of crossover relative to the frequency of the endpoint. For example, if the death rates in the control and treatment groups are 50% and 25%, respectively, crossover of 3% is unlikely to affect the outcome. On the other hand, if the death rates are 8% and 5.5%, respectively, as was the case in the Coronary Artery Surgery Study (CASS) [3], a crossover rate of 25% may seriously distort the results.

Adherence to the experimental protocol is a fundamental principle of research. Large-scale uncontrolled crossover can invalidate any study, no matter what method of data analysis is used.

Meaning of Statistical Power

There is a commonly held but erroneous belief that the failure to find a statistically significant difference between treatment and control may be taken as adequate proof that the treatment is no better (or no worse) than control. This is not so: it is not unusual for a truly superior (or inferior) treatment to fail to produce a statistically significant difference. Such an occurrence is known as a type II error.

In testing a null hypothesis of no treatment difference, we either reject or fail to reject the null hypothesis, but we do not "accept" or "prove" the null hypothesis. The failure to reach statistical significance may be merely the result of an inadequate sample size, which may be expected to lead to a high type II error rate. For example, a randomized study of surgical versus medical treatment of a certain condition may demonstrate an improvement with operation, but the difference may not reach statistical significance. This finding alone does not justify the conclusion that medical treatment will produce the same outcome as surgical treatment, or that operation is "unnecessary" for that condition. Such conclusions are warranted only if the trial were so large and unbiased that small but important treatment differences could be detected with high probability.

The statistical power of a study is equal to one minus the probability of type II error, so that the greater the power, the less likely a type II error. Statistical power has been likened to the resolving power or a microscope: if two points are very close together, a more powerful microscope will be needed to distinguish the distance between them [4]. Conversely, if the two points are far apart, a less powerful microscope will still detect the difference.

In a clinical trial, the statistical power is closely related to both the magnitude of the difference between the groups and to the sample size. Smaller differences will require larger sample sizes to avoid a type II error.

What is an acceptable level of statistical power? There is no one answer to this question. Many studies are planned to achieve a power of 0.80 or greater, meaning that a real difference would be missed no more than 20% of the time. The decision should consider the costs and consequences of missing a real difference of given magnitude. In the CASS randomized study, for example, the 5-year mortality in the medically assigned group was estimated at 8.0% versus 5.5% in the surgically assigned group. Although the difference is only 2.5%, this amounts to a saving of 25,000 lives per one million patients at risk. In a highly prevalent condition, such as coronary artery disease, even a small improvement in outcome may have a large impact on public health, so a higher statistical power (such as 0.90) is desirable. The statistical power of CASS was only 0.33, meaning that a real difference of this magnitude would be missed 67 times out of 100 [5].

Effect of Crossover on Statistical Power

The primary goal of a randomized study is to estimate the true response rates of both treatment and control and to determine the significance of observed differences. When

Table 1. Example Using Crude Mortalities

Variable	Control Group	Treatment Group
No. of patients	400	400
No. of deaths	40	20
Death rate	0.10	0.05
No. of crossovers	100	0
Crossover rate	0.25	0
Observed deaths in crossovers	2	
No. of non-crossovers	300	
Observed deaths in noncrossovers	38	
Calculation for the hypothetical study		
Death rate in crossovers	0.02	(2/100)
Death rate in compliant controls	0.127	(38/300)

	If no crossover were permitted		
Possible values for true death rate in crossovers	Expected No. of Deaths in Crossovers	Expected Total Deaths	Expected Death Rate
0.127	13	13 + 38 = 51	0.153
0.20	20	20 + 38 = 58	0.145
0.25	25	25 + 38 = 63	0.158
0.33	33	33 + 38 = 71	0.178
0.50	50	50 + 38 = 88	0.220

patients in the control group cross over and undergo surgical treatment, it becomes impossible to determine the true response rate to the control therapy. It is possible, however, to derive an estimate of the expected outcome had all the controls remained compliant, as follows. This is one way to determine the diminution in resolving power as a result of the intention-to-treat analysis.

Assume a hypothetical study with 400 patients in the control group (Table 1). The observed death rate is 0.10 (40/400). The crossover rate is 0.25 (100/400). There are two deaths among the crossovers (2/100 = 0.02) and 38 deaths among the compliant controls (38/300 = 0.127).

Under the null hypothesis of no difference between treatment and control, the death rate with either treatment or control is the same, so if all patients were at constant risk of mortality, crossing over should not affect the outcome. The expected death rate for the crossovers, in this case, should be the same as that of the compliant controls, or 0.127. Therefore, one would expect 100 × 0.127, or 13 deaths among the crossovers. The expected death rate for all the controls would be the 38 observed deaths plus the 13 expected deaths, or 51/400 = 0.128, if no crossover were permitted.

However, this estimate of expected outcome is conservative, as the usual reason for crossover is that these patients have become more symptomatic and are therefore more likely to die than the compliant controls. Higher estimates of the death rate in crossovers may be applied to derive more realistic estimates of the expected death rate for the entire control group, assuming no crossover were permitted.

In the Appendix, we present a simple mathematical model for a randomized clinical trial comparing a surgical treatment group with a medical control group, with crossover occurring from the control group to operation. The model is used to generate power curves under various assumptions of mortality rate, sample size, and extent of crossover. This model is a simple binomial model; when trial data are analyzed by actuarial methods, appropriate calculations should be used.

Figure 1 shows the power curves plotted against crossover for a study with 400 patients in each group, a death rate of 0.05 in the treatment group, and death rates of 0.075, 0.10, 0.125, and 0.15 in the control group. Curve C shows an example of the effect of crossover on statistical power: If there were no crossover and the death rate in the compliant controls were 0.10, the power would be about 0.77 (ie, the risk of type II error would be 0.23). When crossover increases to 0.25, the power decreases to

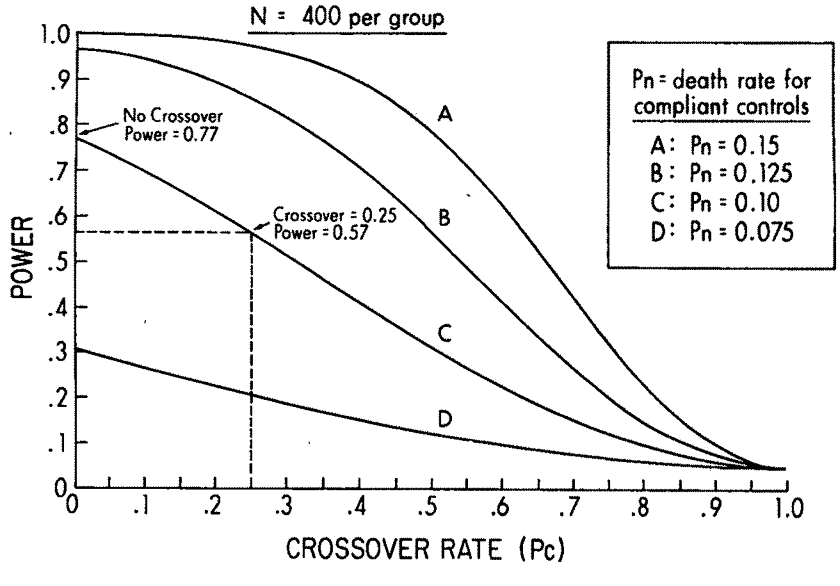

Fig 1. Power to reject the null hypothesis (H_0: $p_1 = p_0$) at $\alpha = 0.05$ and N = 400 per group. [Where $p_0 = p_c \times p_1 + (1-p_c) \times p_n$. See Appendix.] If the surgical death rate (p_1) is 0.05 and the control death rate (p_n) is 0.10 (curve C), with no crossover, the statistical power is 0.77. With a crossover rate of 0.25, the statistical power decreases to 0.57. If operation decreased mortality from 10% to 5%, this difference would be missed 23% of the time with no crossover and 43% of the time with 25% crossover.

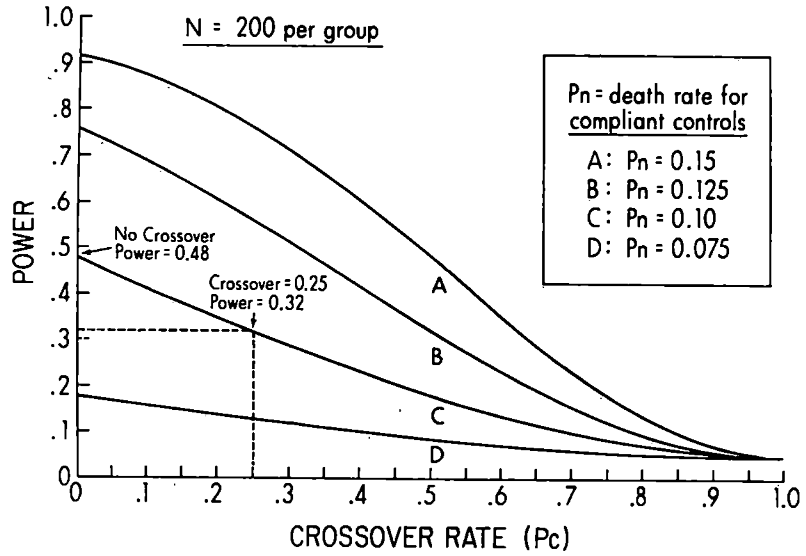

Fig 2. Same as Figure 1, but N = 200 per group. The statistical power decreases with smaller sample size: with no crossover, the statistical power is 0.48. With a crossover rate of 0.25, the power decreases to 0.32. Therefore, if operation decreased mortality from 10% to 5%, this improvement would be missed 52% of the time with no crossover and 68% of the time with 25% crossover.

0.57 (raising the risk of type II error to 0.43). This means that if the death rate of 10% in the medical group were cut in half by operation (to 5%), with no crossover the difference would be found "not statistically significant" 24 times out of 100. With crossover of 25%, this difference would be missed 43 times out of 100.

The effect of sample size on statistical power is shown in Figure 2. In this case, the death rates are the same as in Figure 1, but the sample size is 200 per group: curve C starts at a much lower power, 0.48 with no crossover, and rapidly decreases to 0.32 with 25% crossover. Therefore, an actual decrease in mortality from 10% to 5% would be missed 52% of the time with no crossover and 68% of the time with 25% crossover.

Contrast this with Figure 3, in which a sample size of 800 per group is associated with a power of 0.97, which decreases to 0.85 with 25% crossover. A true decrease in mortality would be missed only 3% of the time with no crossover and only 15% of the time with 25% crossover.

The sample sizes for three major randomized studies of coronary artery operation ranged from 332 to 395 (Table 2). Most studies report results before complete follow-up is available, so the sample size of the "5-year result" is often much smaller than the initial sample size, with a corresponding decrease in statistical power (eg, CASS started with 390 patients in each group, but reported 5-year follow-up for only 280 and 288 patients in the medically and surgically assigned groups, respectively [3]). Therefore, one should be careful to evaluate the statistical power based on the appropriate sample size.

Comment

An understanding of the importance of statistical power is essential in the interpretation of randomized studies. When the statistical power is low, one should not attempt to draw conclusions from a difference that is not statistically significant. Many observers erroneously conclude

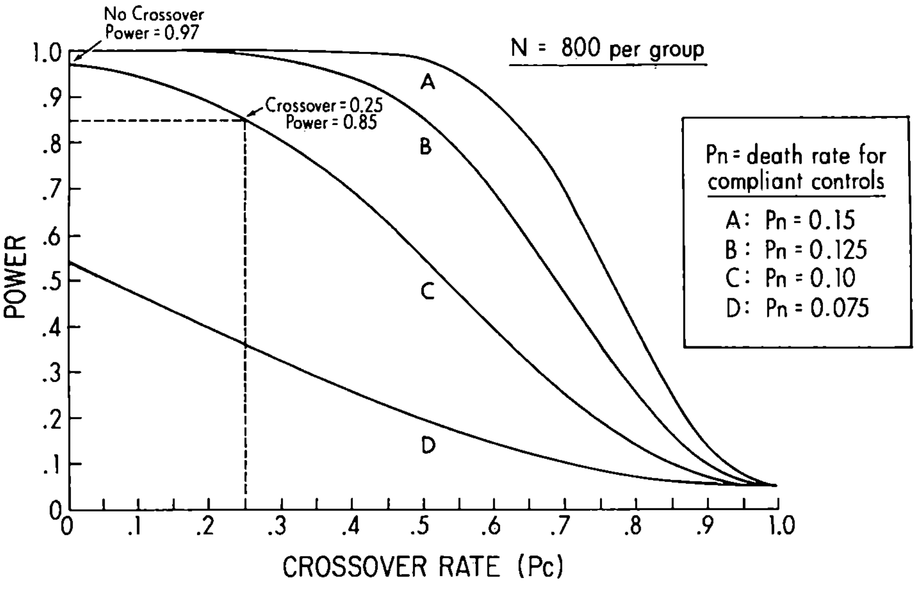

Fig 3. Same as Figures 1 and 2, but N = 800 per group. With no crossover, the power is 0.97; with a crossover rate of 0.25, the power decreases to 0.85. The improvement with operation would be missed only 3% of the time with no crossover and only 15% of the time with 25% crossover.

Table 2. Sample Sizes of Randomized Studies of Coronary Artery Surgery

Study	Initial		5-Year	
	Medical	Surgical	Medical	Surgical
CASS [3]	390	390	280	288
European [6]	373	394	168	215
VA study [7]	354	332	104	114[a]

[a] Sample size at 4 years.

CASS = Coronary Artery Surgery Study; VA = Veterans Administration.

that a difference that fails to reach statistical significance is nonexistent. Still others mistakenly infer that such a difference is of no practical significance [8].

Small sample size is a common cause of inadequate statistical power. Freiman and associates [9] showed that 67 of 71 "negative" randomized trials (94%) suffered from inadequate statistical power. They concluded that many of the therapies labeled as "no different from control" had not received a fair test. Large-scale crossover analyzed under the intention-to-treat principle further degrades statistical power. The Coronary Artery Surgery Study, for example, had a statistical power of only 0.33, meaning that, if CASS were repeated 100 times, a real difference as great as that observed would fail to reach statistical significance 67 times out of 100 [5]. Therefore, it is not sufficient for a randomized study to report that a difference failed to reach statistical significance; the statistical power must also be stated.

Large-scale crossover in randomized studies remains a vexing problem. Although the importance of sample size and statistical power in clinical studies is well known, the effect of crossover on statistical power is rarely addressed. All studies involving long-term follow-up are subject to the effects of such crossover. Both CASS and the European Coronary Surgery Study had crossover of about 25% at 5 years [3, 6]. Crossover in the Veterans Administration Cooperative Study reached 38% by 11 years [10].

The power curves shown in Figures 1 through 3 demonstrate that crossover may have a profound effect on statistical power. This effect is even more pronounced in studies using actuarial methods, as the number of patients followed for 5 years is invariably smaller than the number entering the study. The result is a further decrease in statistical power.

Crossover in randomized studies is so commonplace that it must be anticipated when a study is designed. Using the methods described, investigators may plan an adequate sample size to reduce the likelihood of type II error and avoid the mistake of labeling a truly valuable treatment as no better than control. As Feinstein [11] stated:

> . . . [W]e commit neither sacrilege nor disloyalty by recognizing that randomized trials cannot always be done, that they do not always yield unequivocal answers when done, and that alternative scientific methods must be developed to get satisfactory answers to questions for which randomized studies are either impossible or inadequate.

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Appendix

Calculation of Power Curves

Let p_1 denote true mortality rate with operation, assuming the value 0.05. Each chart assumes sample size N per group, where $N = 200, 400$, or 800. Let the observed death rate in the surgical arm be denoted by p_1. For simplicity, we assume that crossovers occur only in the medical group, in numbers N_c that are binomially distributed with crossover rate p_c, $N_c \sim \mathrm{Bin}(N, p_c)$. Given N_c crossovers, we assume that the number of deaths X_c in the crossover group occur at rate p_1 and binomially distributed, $X_c \sim \mathrm{Bin}(N_c, p_1)$, whereas in the noncrossovers, X_n deaths occur at rate p_n and are binomially distributed, $X_n \sim \mathrm{Bin}(N - N_c, p_n)$. The assumption that p_1 equals the death rate in the crossovers is conservative, given that in CASS [3] the actual death rate among crossovers receiving operation was much lower than p_1.

In the intention-to-treat analysis, the medical arm death rate is given by $p_0 = (X_c + X_n)/N$ with expected rate $p_0 = p_c \cdot p_1 + (1 - p_c) \cdot p_n$, and variance is given by $(p_c p_1[1 - p_1] + [1 - p_c]p_n[1 - p_n] + p_c[1 - p_c]\,[p_1 - p_n]^2)/N$. In the variance expression, the first two terms represent the average binomial variation in deaths at rates p_1 or p_n, and the third term represents binomial variation in the number of crossovers N_c.

Under the null hypothesis H_0: $p_1 = p_n = p_0$, the usual two-sided level α test for significance of the difference in mortality rates is valid in the intention-to-treat analysis, and it rejects H_0 when

$$|p_1 - p_0| > c_{\alpha/2} \sqrt{[2\bar{p}(1 - \bar{p})/N]},$$

where $c_{\alpha/2} = 1.96$ for $\alpha = 0.05$ and $p = ½ (p_0 + p_1)$. (We have ignored the ½-continuity correction for simplicity. It should be incorporated in executing the hypothesis test in practice, and would reduce the reported power figures slightly.)

Under the alternative hypothesis H_1: $p_1 < p_0 < p_n$, the power of the test may be approximated by the formula

$$\text{Power} = \Phi[\sqrt{N} \cdot (a - b) / \sqrt{v}] + \Phi[\sqrt{N} \cdot (-a - b)/\sqrt{v}],$$

where $\Phi(x)$ = the standard normal cumulative distribution function, $a = (p_n - p_1)(1 - p_c)$, $b = c_{\alpha/2} \cdot \sqrt{[2p^*(1 - p^*)/N]}$, where $p^* = ½(p_0 + p_1) = ½[p_cp_1 + (1 - p_c)p_n + p_1]$, and

$$v = p_1q_1 + p_cp_1q_1 + (1 - p_c)p_nq_n + p_c(1 - p_c)(p_1 - p_n)^2.$$

Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 3, 1991. The closing date for registration is August 1, 1990.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

A candidate applying for admission to the certifying examination must fulfill all the requirements of the board in force at the time the application is received.

Please address all communications to the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Appraisal of Resection and End-to-End Anastomosis for Repair of Coarctation of the Aorta in Infancy: Preference for Resection

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Henk J. J. van Lier, MSc, and Leon K. Lacquet, MD

Departments of Thoracic and Cardiac Surgery, Pediatric Cardiology, and Statistical Consultation, Academic Hospital Nijmegen, Nijmegen, the Netherlands

Between 1973 and 1987, 70 consecutive infants underwent repair of coarctation of the aorta. Age at operation was 80.0 ± 77 days (mean ± standard deviation); mean weight was 3.0 ± 0.5 kg. Isolated coarctation was present in 25 patients (group 1); in 19 patients coarctation existed in association with ventricular septal defect (group 2); and in 26 patients coarctation was associated with major intracardiac defects (group 3). Subclavian flap angioplasty was performed in 19 patients and resection and end-to-end anastomosis in 51 patients. Hospital mortality was not significantly different between subclavian flap angioplasty (11%) and resection and end-to-end anastomosis (24%). Freedom from reintervention for recoarctation after 5 years was 87% in the subclavian flap angioplasty group and 95% in the group having resection and end-to-end anastomosis. Actuarial survival at 5 years was 100% for group 1, 73% for group 2, and 28% for group 3. In the subclavian flap angioplasty group, we observed detrimental effects of the sacrifice of the left subclavian artery: 1 patient had a 2.5-cm shortening of the left upper arm, and 5 others complained of claudication in the left upper limb during strenuous exercise. As no major advantage in terms of mortality and recoarctation to either technique of coarctation repair was found, and as subclavian flap angioplasty carries the possible disadvantage of late contracture of isthmic ductal tissue and possible detrimental effects on the left upper limb, resection and end-to-end anastomosis is recommended.

(Ann Thorac Surg 1989;48:496–502)

Traditionally, coarctation of the aorta requiring operation was managed by resection and end-to-end anastomosis (RETE) at our institution. We started to perform the subclavian flap angioplasty (SFA) [1] in 1980 after it had become widespread in the late 1970s. Although reported results have indicated a favorable outcome with SFA [2], this may have been facilitated in part by coincidence of development of the technique with revolutionary changes in preoperative management, particularly the use of prostaglandin E_1 [3], and refinement of microvascular techniques. During the last few years, there has been a slight trend toward reappraisal of RETE because of reported higher rates of early recurrence of coarctation after SFA in patients less than 3 months of age [4, 5]. These findings may reflect residual ductal tissue left in place with SFA, as indicated in various morphological studies [6, 7]. Because we believe the possibility of left arm growth disorders resulting from sacrifice of the left subclavian artery is an additional potential hazard attending this procedure, a retrospective analysis of long-term results including assessment of left upper limb function after SFA is warranted.

Material and Methods

Between 1973 and 1987, 70 consecutive infants aged 80 ± 77 days (mean ± standard deviation) underwent repair of coarctation of the aorta at our institution by either RETE (51 patients) or SFA (19 patients). There were 31 girls and 39 boys. Mean weight at operation was 3.0 ± 0.5 kg. Twenty-three of the 51 RETE procedures were performed between 1973 and 1980 and 28 between 1980 and 1987, whereas the 19 SFA procedures were performed between 1980 and 1987. A patent ductus arteriosus was present in 74% of patients.

The patients were classified into three categories depending on the associated anomalies: group 1 (n = 25), isolated coarctation without major intracardiac anomalies; group 2 (n = 19), coarctation associated with ventricular septal defect; and group 3 (n = 26), coarctation in association with complex intracardiac defects other than simple ventricular septal defect. Various anomalies were present and are listed in Table 1. The diagnosis was suspected clinically and confirmed by two-dimensional echocardiography. Cardiac catheterization was performed only if echocardiography was inconclusive and if additional intracardiac anomalies were suspected. Routinely, catheterization included ventriculography, aortography, pressure determinations, and oximetry.

The indication for operation was congestive heart failure unresponsive to medical treatment in 66% of the

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Table 1. Associated Lesions in the 26 Group 3 Patients

Lesion	No. of Patients
Aortic valve stenosis	7
d-TGA, VSD	5
Atrioventricular canal	3
Subaortic stenosis	2
Aortic and mitral valve stenosis	2
l-TGA, VSD	1
Single ventricle	1
d-TGA, single ventricle	1
Aortopulmonary window	1
Hypoplastic left heart	1
d-TGA, VSD, dextrocardia	1
Double-outlet right ventricle	1

d-TGA = dextrotransposition of great arteries; l-TGA = levotransposition of great arteries; VSD = ventricular septal defect.

patients, severe hypertension (equal to or exceeding systolic pressure of 150 mm Hg) in 16%, and a combination of both in 18%. Medical management of congestive heart failure consisted of digitalis, diuretics, and inotropic medication if necessary. Since 1980, prostaglandin E_1 has been used to maintain ductal patency in neonates with critical coarctation.

Associated noncardiac complications were present preoperatively in 3 infants who sustained cerebrovascular accidents secondary to hypertensive crises with blood pressures exceeding 200 mm Hg. In 2 of these patients neurological symptoms were transient, but in the other, chronic paresis of the left foot developed.

Follow-up

Follow-up data were available through our clinical records of outpatient visits. The patients were seen at 3-month intervals during the first year after operation and yearly thereafter. Forty-six (98%) of 47 hospital survivors were followed for 4.7 ± 3.5 years (range, 0.1 year to 13.2 years). Parents and children were questioned about symptoms relating to left upper limb ischemia or subclavian steal syndrome. In addition, inquiries were made about differences in temperature or size between the upper limbs.

Resting systolic blood pressures were measured in both brachial arteries and the right femoral artery using a sphygmomanometer and a unidirectional 8-MHz Doppler ultrasound velocity detector. Blood pressure cuffs were placed on both upper arms and the right thigh with bladder size chosen to cover approximately 75% of limb length [8]. If there was local stenosis of the right femoral artery, the resting blood pressure of the left femoral artery was determined. The brachial–femoral artery pressure gradient was calculated by subtracting femoral systolic pressure from right brachial systolic pressure. Blood pressure measurements were made preoperatively, in the immediate postoperative period (within 72 hours after operation), and at late follow-up.

Surgical Technique

Through a left posterolateral thoracotomy in the fourth intercostal space, the aorta is mobilized with dissection of the transverse aortic arch, the left subclavian artery, and the upper half of the descending thoracic aorta. Intercostal arteries are sacrificed only as required for mobilization of the descending aorta. The ductus arteriosus is divided and oversewn, or the ligamentum arteriosum is divided.

The proximal vascular occlusive clamp is placed across the aortic arch and the left subclavian artery so that patency of the left common carotid artery is maintained. The distal clamp is placed across the upper descending aorta distal to the coarctation. The stenotic isthmic segment is completely excised as well as any residual ductal tissue. In patients with tubular hypoplasia, the proximal clamp is positioned just beyond the innominate artery, thus occluding the left common carotid artery.

Subsequently, the resection is extended to the origin of the left subclavian artery, and the inferior aspect of the aortic arch is incised in a cephalad direction to achieve a maximally wide anastomotic diameter (Fig 1). If necessary, the distal aorta is trimmed obliquely and a matching anastomosis is created (Fig 2). The anastomosis is performed either with interrupted sutures placed circumferentially, with a running suture on the posterior aspect and interrupted sutures anteriorly, or a circumferentially running suture (6-0 polypropylene). Alternatively, since 1981, a running 6-0 polydioxanone suture has been used.

If there is residual stenosis of the distal transverse aortic arch due to the proximal extent of the hypoplastic aortic segment, the intraluminal diameter is increased by incision and anastomosis of the left common carotid and left subclavian arteries [9]. When the aortic arch between the

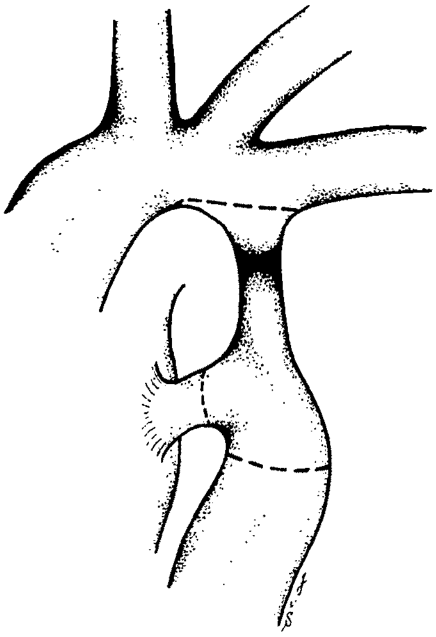

Fig 1. Incision lines in the transverse aortic arch and the upper descending aorta to allow the widest possible aortic diameter for anastomosis.

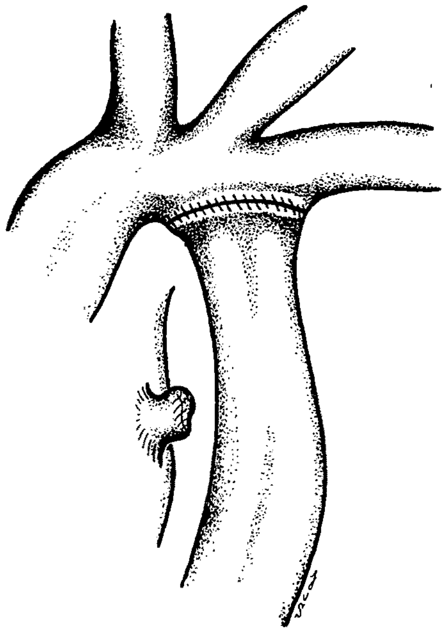

Fig 2. End-to-end anastomosis after resection of the coarctation. The ductus arteriosus is divided and oversewn.

left common carotid and left subclavian arteries is not only hypoplastic but also elongated, the concavity between the hypoplastic arch and left subclavian artery is incised, and the two are sutured together [10].

When SFA is performed, the distal transverse aortic arch, isthmus, left subclavian artery, proximal descending aorta, and ductus arteriosus are mobilized. Clamps are placed across the transverse aortic arch and the descending aorta well beyond the level of the coarctation. Occasionally it may be necessary to sacrifice one or two pairs of intercostal arteries above the level of the distal clamp. The ductus arteriosus is divided and oversewn. The left subclavian artery is ligated and transected distally at the thoracic outlet beyond the ligated vertebral artery. A longitudinal incision is carried out along the lateral wall of the left subclavian artery toward the aorta, through the stenotic aortic isthmus, and well beyond onto the lateral wall of the descending aorta to avoid residual narrowing at the site of (or just distal to) the coarctation.

In all instances, the posterior diaphragm is excised as completely as possible. Even if the flap is wide, the distal corners are but scarcely trimmed so that the flap is as wide as possible. Subsequently, a 6-0 polypropylene or polydioxanone suture is passed through the apex of the flap and through the lowest point of incision in the descending aorta. Finally, two running suture lines are started at the proximal end of the flap and extended to the apex.

Statistical Analysis

Categorical data were arranged in contingency tables, and the Fisher exact test or the χ^2 test was used for significance. Proportions are accompanied by their 95% confidence limits (CL). The data on survival and recurrence rates were analyzed by the actuarial method of Kaplan and Meier. Quantitative data were compared with the Kruskal-Wallis test. Statistical significance was reached at a p value equal to or less than 0.05.

Table 2. Hospital Mortality Rate in Relation to Anatomical Category After Coarctation Repair in Infancy

Anatomical Category	Alive	Dead	Total	Hospital Mortality (%)	95% Confidence Limit
Group 1[a,b]	25	0	25	0	0%–14%
Group 2[c]	17	2	19	11	1%–33%
Group 3	14	12	26	46	27%–67%
Total[d]	56	14	70	20	11%–31%

[a] Significance: $p = 0.36$, group 1 versus group 2. [b] Significance: $p = 0.0004$, group 1 versus group 3. [c] Significance: $p = 0.013$, group 2 versus group 3. [d] By χ^2 test, $p < 0.0001$.

Results

Hospital Deaths

The overall operative and hospital mortality (≤30 days) was 20% (14/70) (CL, 11% to 31%). Hospital mortality rates varied by patient group: 0% (0/25) (CL, 0% to 14%) for group 1 (isolated coarctation) and 11% (2/19) (CL, 1% to 33%) for group 2 (coarctation plus ventricular septal defect). The combined mortality for these two groups of 5% (2/44) (CL, 1% to 15%) was significantly different from the mortality of 46% (12/26) (CL, 27% to 67%) for group 3 (coarctation with intracardiac defects other than ventricular septal defect) (Table 2). The average age (± the standard deviation) of the hospital survivors at operation was 92.1 ± 79.5 days compared with 30.2 ± 40.6 days for those who died ($p < 0.0005$). There was no significant difference in hospital mortality rate between the SFA and RETE groups, 11% (2/19) (CL, 1% to 33%) versus 24% (12/51) (CL, 13% to 37%), respectively (Table 3).

The most common cause of hospital death was acute cardiac failure after repair (57%, 8/14) (CL, 29% to 82%). Other causes included pulmonary dysfunction (29%; 4/14) (CL, 8% to 58%) and renal insufficiency (14%, 2/14) (CL, 2% to 43%).

Late Deaths

Among the 56 hospital survivors there have been nine late deaths (three in the SFA group and six in the RETE

Table 3. Hospital Mortality Rate in Relation to Method of Coarctation Repair

Method of Repair	Alive	Dead	Total	Hospital Mortality (%)	95% Confidence Limit	p Value[a]
RETE	39	12	51	24	13%–37%	NS
SFA	17	2	19	11	1%–33%	
Total	56	14	70	20	11%–31%	

[a] The value of p by χ^2 test = 0.38.

NS = not significant; RETE = resection and end-to-end anastomosis; SFA = subclavian flap angioplasty.

Table 4. Additional Procedures in Group 3 Patients

Procedure	No. of Patients	Timing of Repair
PA banding	10	Simultaneous with coarctation repair
Closure of atrioventricular canal	1	Two-stage repair
Aortic valvotomy	1	Two-stage repair
Mitral commissurotomy	1	Two-stage repair
Correction of aortopulmonary window	1	Two-stage repair
Closure of VSD, debanding of PA, arterial switch for d-TGA	1	Two-stage repair
Total	15	

d-TGA = dextrotransposition of great arteries; PA = pulmonary artery; VSD = ventricular septal defect.

group). The actuarial survival at 5 years was 100% for group 1 patients, 73% for group 2 patients (standard error = 10%), and 28% for group 3 patients (standard error = 9%). In all instances death was due to progressive congestive heart failure.

Complications

In 1 patient, a 2.5-cm shortening of the left upper arm developed 6 years after SFA. Five children in the same group, all 5 years of age or older, complained of claudication in the left upper limb during strenuous exercise (in 4 while swimming and in 1 during gymnastics). All patients in the SFA group had diminished or absent pulses in the left upper limb. No correlation was found between systolic blood pressure and pulse volume or symptoms. Paradoxical hypertension in the early postoperative period, defined as sustained systolic and diastolic blood pressures greater than the 95th percentile for age, occurred in 2 patients. Both were treated with infusions of sodium nitroprusside, and normal blood pressures developed during the third and fourth postoperative days.

Subsequent Operative Procedures

Three patients (2 from the SFA group and 1 from the RETE group) successfully underwent transfemoral balloon angioplasty for residual coarctation with gradients exceeding 30 mm Hg between 7 and 15 months after the initial procedure. Another patient required prosthetic patch graft angioplasty 4 months after an SFA procedure. A constriction at the posterior aortic wall opposite the subclavian patch appeared to have narrowed the aorta. The stenosis was relieved by a Gore-Tex patch graft. Additional procedures in group 3 patients are listed in Table 4.

Hypertension

Before coarctation repair, 27 (39%) (CL, 27% to 60%) of the 70 patients were considered hypertensive. At late follow-up, 8 (17%) (CL, 8% to 31%) of 46 survivors were hypertensive. Operative technique had no major influence on the development of late hypertension: after RETE, 5 (16%) (CL, 5% to 33%) of 32 patients were hypertensive and after SFA, 3 (21%) (CL, 5% to 51%) of 14 patients. All 8 patients with hypertension postoperatively had gradients greater than 20 mm Hg between the right brachial and femoral arteries.

Recoarctation

The incidence of persistent and recurrent coarctation (defined as a right brachial–femoral artery pressure gradient equal to or exceeding 20 mm Hg in the immediate postoperative period and at late follow-up after its initial absence) was not significantly different between RETE and SFA patients: respectively, 9% (4/45) (CL, 3% to 21%) versus 17% (3/18) (CL, 4% to 41%) and 16% (5/32) (CL, 5% to 33%) versus 29% (4/14) (CL, 8% to 58%). Freedom from reintervention at 5 years for patients who underwent RETE was 95% (standard error = 3.5%) compared with 87% (standard error = 9%) for patients in the SFA group

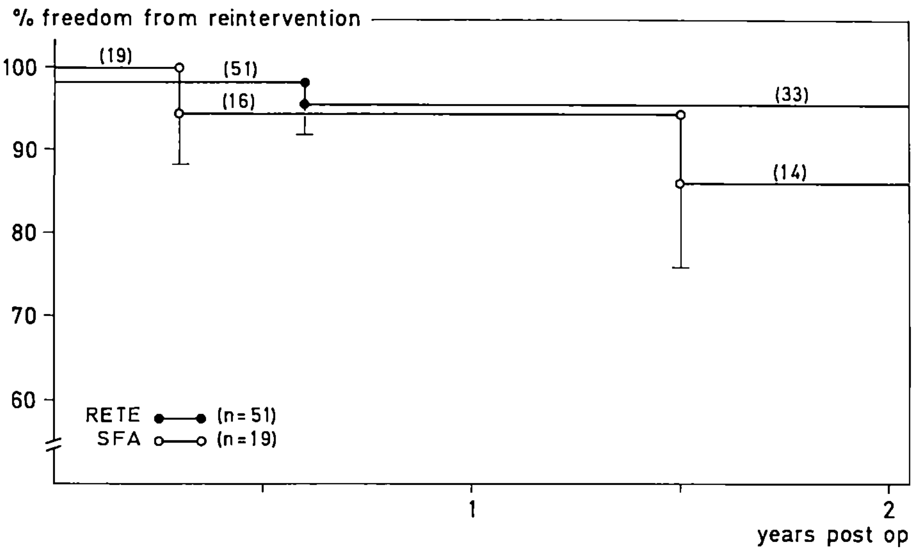

Fig 3. Freedom from reintervention according to type of repair (Kaplan-Meier method). (RETE = *resection and end-to-end anastomosis;* SFA = *subclavian flap angioplasty.)*

(Fig 3). Type of suture material (polypropylene versus polydioxanone) and technique (interrupted sutures circumferentially versus a running suture on the posterior aspect and interrupted sutures anteriorly versus a circumferentially running suture) did not appear to have a significant influence on recurrence of coarctation.

Comment

Since the introduction of RETE for coarctation of the aorta four decades ago [11], there has been controversy over the proper management of this common congenital defect in children. High rates of recurrence of coarctation in infants treated by RETE were attributed to inadequate resection of the coarctation as well as disproportional growth of a circumferential suture line [12, 13].

To overcome the problem of residual coarctation and the high early mortality rates, patch graft angioplasty [14] and SFA [1] were developed. Both are expeditious procedures and avoid extensive dissection as well as potential constriction from a suture line. Patch graft angioplasty appears to have a lifelong threat of aneurysm formation opposite the graft [15]. A lower operative mortality and a lower recurrence rate have been reported for SFA than for RETE and patch graft angioplasty [2]. The high success rate of SFA may partially reflect the later time frame during which it was performed with advanced microvascular techniques and materials, the simultaneous introduction of prostaglandin E_1 for preoperative management [3], and improved postoperative care.

In the recent literature there is a slight trend toward reappraisal of RETE [4, 5, 12, 13] or extended RETE [16]. Reasons for this change in attitude can be attributed to the relatively high recurrence rate of coarctation in children after SFA done when they were less than 3 months of age [4] and to accumulating evidence from arterial switch operations that circumferential suture lines per se may not preclude satisfactory growth of the anastomosis [17]. We detected a recurrent gradient after its initial absence in 4 (29%) of 14 patients who underwent SFA when they were younger than 3 months compared with 5 (16%) of 32 in the RETE group. However, 4 of the latter were operated on in earlier years when the suture techniques were less refined. Angiographically a prominent diaphragm opposite the subclavian flap could be detected in 3 patients with a recurrent gradient in the SFA group. They subsequently underwent successful reintervention by balloon angioplasty or Gore-Tex patch graft angioplasty. The fourth patient will continue to be evaluated and possibly will undergo reintervention in the future. Our 29% rate of recurrence of coarctation in infants undergoing SFA at an age of less than 3 months is similar to the experience in other centers [4, 5].

Several authors [6, 7] have emphasized the decisive role that ductal tissue can have in the evolution of the isthmus in the neonatal stage. Besides this hypothesis of Skoda [18], strong circumstantial evidence has been presented in support of a hemodynamic pathogenesis of coarctation [19]. In the presence of intracardiac congenital lesions with left-to-right shunting, there may be a synergistic relationship between both mechanisms: augmented flow through the ductus arteriosus with concomitant diminished anterograde flow through the aortic arch in embryonic and fetal life may lead to a well-developed ductus arteriosus contrasting with an underdeveloped aortic arch.

This disproportional growth of both structures may facilitate proliferation of ductal tissue in a high-flow, and consequently low-pressure, zone. As highest flow occurs at the lateroposterior side of the entrance of the isthmus to the common channel formed by the ductus arteriosus and the descending aorta, ductal tissue will almost invariably be found at this location. Thus, when the aorta is viewed from an anterior position, it is suspended as a diaphragmatic structure mainly from the lateroposterior aortic wall and facing the descending aorta [6, 7]. It may partially or completely encircle the orifice of the isthmus, thus causing contracture at the isthmic level at the time of ductal closure, especially when ductal intimal cushions are present in the aorta [7]. This is reflected in the high association of preductal coarctation with isthmic or tubular hypoplasia, or both, in children with these fetal flow patterns [6].

On the other hand, in a histological study by Elzenga [20] of aortic arches from postmortem specimens with an obstructive aortic arch anomaly, less than half of paraductal coarctations contained ductal tissue in the diaphragm opposite the ductus arteriosus. A similar study of specimens from infants with hypoplastic left heart syndrome even showed complete absence of ductal tissue in the ridge of the paraductal coarctations [20]. Paraductal coarctation seems to occur in those arches that, in embryonic and fetal life, apparently have received part of their flow in a retrograde fashion from the ductus arteriosus. Hence, paraductal coarctation can be regarded as a branch-point of the ductus arteriosus [21]. Depending on the timing of the flow disturbance and the magnitude of anterograde and retrograde flow passing the watershed formed at this branch-point, stimuli may be provided to proliferation of ductal tissue at the downstream (facing the descending aorta) or upstream (facing the aortic arch) sides, or both, of the diaphragmatic ridge opposite the ductus arteriosus. As hypoplastic left heart syndrome is thought to develop relatively late in embryonic life and as the combined ventricular output is markedly decreased, it appears to rarely present with ductal tissue in the associated (paraductal) coarctation.

In this perspective, the distribution of ductal tissue in the diaphragms of the two subtypes of coarctation suggests that they represent extremes in a spectrum of anomalies of the aortic isthmus and arch rather than two different entities. Generally, our hypothesis is that increased flow through the embryonic and fetal ductus arteriosus can lead to excessive extension of ductal tissue into the aortic wall. Depending on the direction and magnitude of flow, proliferation of ductal tissue may be anterograde or retrograde, or both, whereas in a considerable number of cases of paraductal coarctation, usually secondary to a markedly decreased ventricular output, ductal tissue in the aortic wall may be absent. Herewith, a synthesis of the theories of Skoda [18], Rudolph and colleagues [19], and Hutchins [21] seems to be reached.

The implication of these data is that in infants, particularly in those less than 3 months of age, all ductal tissue should be removed to prevent recurrence of coarctation. This pertains especially to those situations in which a coarctation of the preductal type, frequently with marked isthmic or tubular hypoplasia, or both, is found. Theoretically, in the presence of isolated paraductal coarctation, both RETE and SFA should yield hemodynamically satisfactory results. However, because the presence of an externally visible circumscribed paraductal indentation of the aorta is not always, in our experience, a reflection of the intraluminal findings, we prefer to carefully inspect the lumen of the isthmus and the proximal descending aorta. In our opinion this is best accomplished by a transverse section through the aortic wall, as performed in the RETE technique. Thus, all ductal tissue can be included in the excised aortic segment.

The 95% 5-year probability of freedom from recoarctation using RETE in this series compares favorably with that in other reports in the literature [4, 5]. Previously, disadvantages related to the circumferential suture line after RETE may have been overemphasized. High recoarctation rates in the past have been the result not only of less-refined suture techniques, but also of the fear to remove too long a segment of the aortic isthmus. As Brom [22] pointed out more than two decades ago, after sufficient mobilization, an end-to-end anastomosis without excessive tension can always be carried out. This is the case even after resection of a long segment because of the extreme elasticity of the aorta in the pediatric age group.

Detrimental effects of the sacrifice of the left subclavian artery were noted in 6 patients. One patient had left upper arm shortening of 2.5 cm. Five others complained of claudication in the left upper limb during strenuous exercise. This observation contrasts with the conclusions in a study [23] published in 1983 that the excellent results with SFA are not compromised by the minor effects of ligating the left subclavian artery in infancy. Other studies [24, 25] found that interruption of the subclavian artery in growing individuals usually causes a substantial diminution in the longitudinal growth of the long bones or diminution in the muscle thickness of the corresponding arm, or both.

A third drawback of SFA is inherent in the need to excise completely the diaphragm of the coarctation to remove the ductal tissue. This maneuver can leave a rough surface on the internal part of the aortic wall, with increased risk of thrombosis, granulation, retraction, or restenosis. Conversely, if resection of the diaphragm is too radical, a weakened area can be created with potential for development of a pseudoaneurysm [15].

In conclusion, this experience with repair of coarctation of the aorta in infants did not show superiority of SFA to RETE in terms of a reduced mortality rate, despite partially different ancillary therapeutic methods during two subsequent time frames. On the other hand, we observed the development of a recurrent gradient in 29% of the patients after SFA performed when they were less than 3 months of age, a development most likely caused by contracture of ductal tissue around the aortic isthmic orifice. Detrimental sequelae to the sacrifice of the left subclavian artery, ie, differential left arm growth rate and claudication in the left upper limb during exercise, were considerable. We believe that complete excision of the coarcted segment is preferable to prevent the propensity of the ductal tissue in situ to continue to constrict the isthmus. Our results show that creation of a satisfactory end-to-end anastomosis with preservation of growth potential of the aorta should be considered a hemodynamically adequate repair.

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REVIEW OF RECENT BOOKS

Thoracic Oncology

Edited by Jack A. Roth, John C. Ruckdeschel, and
Thomas H. Weisenburger
Philadelphia: W.B. Saunders, 1989
740 pp, illustrated, $90.00

Reviewed by Thomas W. Shields, MD

Doctors Roth, Ruckdeschel, and Weisenburger are to be congratulated for the excellent text *Thoracic Oncology*, in which they have presented a very acceptable combined multimodality approach to the cancers occurring in or metastasizing to the thorax. Not only have they addressed the clinical presentation and management of the various tumors, but they have also presented a knowledgeable account of cellular and molecular biology and immunology of the various major tumors. The clinician must be aware of and have a better understanding of the contributions these areas have added and will continue to add to the diagnostic and therapeutic techniques now currently available in the management of these serious diseases.

As one would suspect, the various features of cancer of the lung and esophagus comprise over half of the volume. These chapters are expertly done and all are well referenced. The chapter on the treatment of small cell lung cancer by Feld and associates is, in my opinion, outstanding. The section on malignancies of the mediastinum is satisfactory, but its organization is unusual and the depth of coverage is variable in the nine chapters in this section. The lack of a surgical co-author for the chapter on malignancies of the thymus is difficult to understand.

Sections on malignancies of the chest wall and pleura, pulmonary metastases, supportive care, and prospects for the future are well done and contain important information for the members of the multidisciplinary team managing these unfortunate patients.

This book would be most worthwhile for the general thoracic surgeon in practice, and especially for the cardiothoracic surgeon (or even the general surgeon) who is called on to assume the major role or, on occasion, to assist in the management of many of these patients. It is important that he or she know the available options the radiation and medical oncologists have to offer. On the other hand, except for the few strictly surgical chapters, this book would appear to be less valuable for the young thoracic surgeon in training because the surgical aspects of many of the disease processes presented are taken for granted and somewhat oversimplified. At times in reviewing the volume, a "Goldilockian" feeling of visiting the three bears became somewhat pervasive: the bowl of surgical porridge was too empty, the bowl of chemotherapy was too full, and that of radiation therapy was just about right.

Despite this mild criticism, the text is overall an important addition to the library of the thoracic oncologist, whether surgeon, radiation therapist, or medical oncologist. It is an excellent addition to one's library for handy reference for authoritative information.

Chicago, Illinois

Delayed Myocardial Metabolic Recovery After Blood Cardioplegia

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Previous studies have demonstrated that both myocardial metabolism and ventricular function were depressed after blood cardioplegic arrest for elective coronary artery bypass grafting. To evaluate the etiology of this metabolic defect, we measured the levels of adenine nucleotides and their precursors in 29 patients undergoing elective coronary revascularization. Myocardial biopsy specimens were obtained at 37°C before cardioplegic arrest, immediately after 74 ± 4 minutes of cardioplegic arrest, and after 30 minutes of reperfusion. Biopsy specimens were analyzed for levels of adenine nucleotides and their precursors by high-performance liquid chromatography. Adenosine triphosphate concentrations decreased with cardioplegic arrest and with reperfusion. Adenosine monophosphate concentrations increased after cardioplegic arrest and remained nearly twice the initial values after reperfusion. The ratio of adenosine monophosphate to adenosine triphosphate doubled after reperfusion, suggesting defective conversion of adenosine monophosphate to adenosine triphosphate. Levels of adenine nucleotide degradation products (adenosine, inosine, and hypoxanthine) increased after cardioplegia and decreased with reperfusion, suggesting a washout of soluble precursors. This study suggests that improvements in myocardial protection should attempt to stimulate mitochondrial energy production and preserve adenine nucleotide precursors.

(Ann Thorac Surg 1989;48:503–7)

Improved methods of myocardial protection may decrease the morbidity and mortality of cardiac operations, particularly for high-risk patients [1–3]. In previous studies, we showed that blood cardioplegia (BCP), particularly with a warm terminal cardioplegic infusion ("hot shot"), improved myocardial metabolic and functional recovery better than crystalloid cardioplegia after elective coronary artery bypass grafting [4–6]. In addition, BCP reduced the risks of urgent revascularization for unstable angina [3]. However, myocardial metabolic [7] and functional [6] recovery were delayed despite apparently adequate blood cardioplegia.

The delayed recovery of ventricular function may have resulted from low postoperative concentrations of adenosine triphosphate (ATP), a consistent finding in our previous studies [4, 5, 7]. This study was intended to determine whether the depressed high-energy phosphate (HEP) concentrations resulted from inadequate mitochondrial energy production [8] or the washout of soluble adenine nucleotide precursors [9].

Material and Methods

Patients

Twenty-nine patients scheduled for elective coronary revascularization agreed to participate in the study and signed a consent form approved by our Institutional Human Subjects Review Committee on July 28, 1987. All patients had stable angina pectoris, preserved left ventricular function (ejection fraction greater than 30% at preoperative cineventriculography), and double-vessel or triple-vessel coronary artery disease.

Surgical Technique

The techniques of anesthesia induction and cardiopulmonary bypass have been described previously [4, 5, 7]. The cardiopulmonary bypass circuit was primed with 2 L of Ringer's lactate to increase arterial lactate concentrations [7]. Moderate hemodilution (hematocrit, 22% to 24%) and moderate systemic hypothermia (nasopharyngeal temperature, 25°C) were used during bypass. Blood cardioplegia was administered with the Buckberg-Shiley Plus System (Shiley, Irvine, CA), which delivered oxygenated blood and a crystalloid solution at a ratio of 2:1. The physical and biochemical composition of the blood cardioplegic solution has been described in previous reports [3–7]. Initially, 1 L of BCP was infused into the aortic root at a pressure of 69 ± 3 mm Hg and a temperature of 6.1° ± 0.5°C, resulting in a cardioplegic flow of 298 ± 32 mL/min. Proximal and distal anastomoses were constructed during a single prolonged cross-clamp period [4–6]. One hundred milliliters of BCP was infused into each vein graft after completion of each distal anastomosis and 400 mL was infused into the aortic root at a pressure of 60 ± 2 mm Hg and a temperature of 6.8° ± 0.7°C, producing a cardioplegic flow of 264 ± 11 mL/min. Approximately 1 L of iced (6°C) Ringer's lactate was applied to the heart with each 400-mL cardioplegic infusion. The left ventricle was vented through the aortic root between cardioplegic infu-

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sions. The left internal mammary artery was used to bypass the left anterior descending coronary artery as the last distal anastomosis. Systemic rewarming was started during construction of the final anastomosis. Before release of the aortic cross-clamp, 500 mL of 37°C BCP was infused into the aortic root at a pressure of 51 ± 4 mm Hg (hot shot) [5]. After cross-clamp release, bypass flow rates were maintained between 2.0 and 2.5 L/min/m^2, and arterial pressures were maintained between 50 and 70 mm Hg.

Measurements

Myocardial temperatures were measured with needle thermistors after each aortic root cardioplegic infusion. The left ventricular temperatures were measured in the distribution of the left anterior descending, circumflex, and right coronary arteries with each cardioplegic infusion, and the lowest value and mean of the temperature measurements were recorded for each patient.

Transmural left ventricular biopsy specimens were obtained with a Tru-Cut biopsy needle (Travenol Laboratories, Deerfield, IL) on cardiopulmonary bypass at 37°C before cross-clamp application, immediately after cross-clamp release, and after 30 minutes of reperfusion. The left ventricular biopsy specimens were obtained from the region subserved by the most stenotic vessel as previously described [4, 5, 7]. Biopsy specimens were not taken from an area of myocardial scarring. The specimens were immersed in liquid nitrogen within 2 seconds and then freeze-dried at −50°C overnight. The dry muscle was separated from any contaminating tissue and weighed. The muscle was homogenized for ten minutes at 4°C with 20 μL/mg tissue of 0.5 M perchloric acid. The sample was centrifuged at 1,260 g for ten minutes, and the supernatant was neutralized with 2 M potassium hydroxide to a pH of 7.6 and then reacidified with 0.1 M perchloric acid to a pH of 6.8. The supernatant was assayed immediately or stored overnight at −80°C.

High-performance liquid chromatography was used to measure the levels of adenine nucleotides and their degradation products. We used a modification of the step-gradient technique described by Hull-Ryde and colleagues [10]. Sample injection was accomplished with a manual injector (model U6K, Waters Associates, Mississanga, Ont, Canada). Step-gradient solvent delivery was performed with a reciprocating pump (Waters Associates, models 501 and 510). The chromatographic column was a Radial-Pak Resolve C18 Column with a 5-μm particle size (Waters Associates) which was operated in a radial compression module (Waters Associates, model RCM 100) at 175 bar. A programmable multiwavelength detector (Waters Associates, model 490) was used to monitor and integrate the peaks at absorbances of 154 and 229 nm.

Solvent flow was initiated isocratically with a 100-mM ammonium phosphate buffer at pH 5.7 for 15 minutes; then the solvent was converted to 40% methanol:water (vol/vol) in a gradient fashion over five minutes using curve 8 of the solvent programmer. The 40% methanol solvent then was infused for an additional ten minutes. Between injections, the column was equilibrated with the ammonium phosphate buffer. An internal standard (2′-O-methyladenosine) was added at a known concentration with each run. Primary and internal standards were used to quantitate the elutants. The system accurately measured uric acid, ATP, inosine monophosphate, adenosine diphosphate (ADP), hypoxanthine, xanthine, adenosine monophosphate (AMP), and inosine (which were measured at 254 nm), and creatinine phosphate (which was measured at 229 nm). The results are expressed as micromoles per gram of dry weight of myocardial muscle. The lower range sensitivity of our high-performance liquid chromatography measurements was approximated from our standard dilutions. The detectable concentration was 100 pmol/L for the adenine nucleotides, 300 pmol/L for xanthine and hypoxanthine, and 300 pmol/L for inosine monophosphate and inosine.

Table 1. Clinical Information

Patients	29
Age (yr)	58 ± 2
NYHA class (II/III/IV)	10/17/2
No. of coronary bypass grafts	
3	12
4	12
5	5
Internal mammary artery grafts	27
Cardiopulmonary bypass time (min)	124 ± 5
Cross-clamp time (min)	74 ± 4
Lowest LV temperature (°C)	8.0 ± 0.3
Mean LV temperature (°C)	12.0 ± 0.4
Highest postoperative CK MB (U/L)	44 ± 4

CK MB = myocardial specific isoenzyme of creatine kinase; LV = left ventricular; NYHA = New York Heart Association.

The following indexes were calculated: total adenine nucleotides (TAN = ATP + ADP + AMP), total adenine nucleotide degradation products (TDP = adenosine + inosine + hypoxanthine), and total adenine nucleotides and degradation products (TNP = ATP + AMP + ADP + adenosine + inosine + hypoxanthine). The energy charge was calculated from the formula EC = (ATP + 0.5 ADP)/(ATP + ADP + AMP) and the level of HEPs was calculated from the formula HEP = ATP + CP.

Statistical Analysis

The Statistical Analysis System programs (SAS Institute, Carey, NC) were used for statistical analysis. An analysis of variance was performed to test the effect of time on tissue metabolites. Duncan's multiple-range test was used to specify differences when analysis of variance was significant ($p < 0.05$). Results are given as the mean and the standard error of the mean.

Results

Intraoperative left ventricular temperatures during cardioplegic arrest (Table 1) were consistently between 10° and 15°C. Postoperatively, no patient died, suffered a

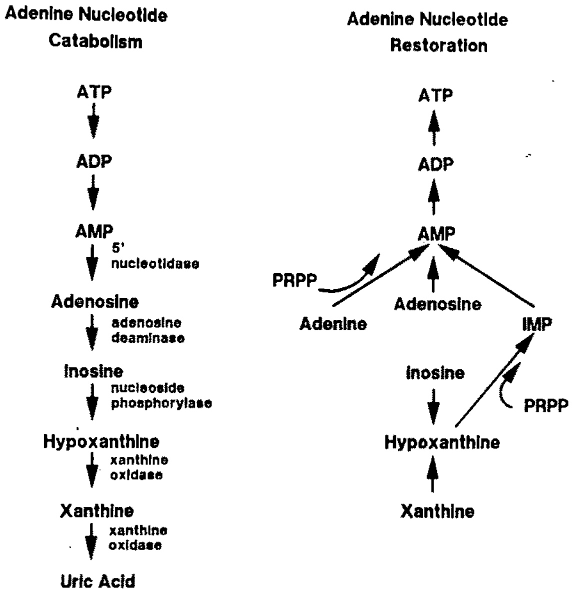

Fig 1. Pathways for adenine nucleotide degradation and restoration. Inosine monophosphate (IMP) *concentrations were not detected in the human myocardium before or after cardioplegic arrest. Therefore, the human heart did not use IMP for either adenine nucleotide degradation or restoration. In addition, xanthine concentrations were not detected, suggesting that xanthine oxidase was not active in the human heart.* (ADP = *adenosine diphosphate;* AMP = *adenosine monophosphate;* ATP = *adenosine triphosphate;* PRPP = *phosphoribosyl pyrophosphate.*)

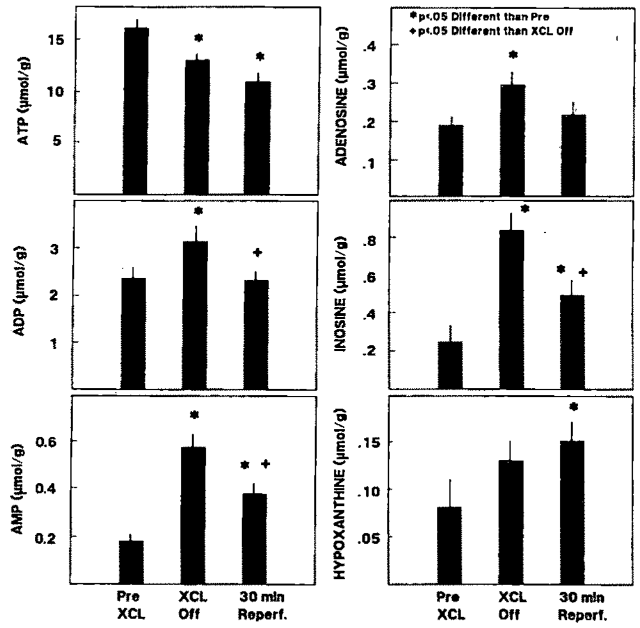

Fig 2. The concentrations of adenine nucleotides (adenosine triphosphate [ATP], adenosine diphosphate [ADP], and adenosine monophosphate [AMP]) (left) and their degradation products (adenosine, inosine, and hypoxanthine) (right). Measurements were made at 37°C on cardiopulmonary bypass before aortic cross-clamping (Pre XCL), *immediately after cross-clamp removal* (XCL Off), *and after 30 minutes of reperfusion.*

perioperative myocardial infarction, or required inotropic support in the intensive care unit.

The products of adenine nucleotide degradation and three pathways for the restoration of AMP are shown in Figure 1. The concentrations of the adenine nucleotides and their degradation products are shown in Figure 2. Adenosine triphosphate concentrations decreased from 15.9 µmol/g of freeze-dried myocardium to 12.8 µmol/g after cardioplegic arrest and then to 10.6 µmol/g after reperfusion. All 29 patients had such a decrease. Adenosine diphosphate concentrations increased from 2.37 to 3.11 µmol/g during cardioplegic arrest and returned to prearrest values after reperfusion. Adenosine monophosphate concentrations increased threefold from 0.18 to 0.58 µmol/g after cardioplegic arrest and returned to 0.38 µmol/g at 30 minutes of reperfusion. Adenosine concentrations nearly doubled from 0.19 to 0.3 µmol/g during cardioplegia but returned to prearrest values after reperfusion. Inosine concentrations increased threefold from 0.25 to 0.83 µmol/g during cardioplegic arrest and were nearly twice the baseline value at 0.49 µmol/g 30 minutes after reperfusion. Hypoxanthine concentrations increased from 0.08 to 0.13 µmol/g during cardioplegic arrest and then remained elevated at 0.15 µmol/g 30 minutes after reperfusion.

Neither inosine monophosphate nor xanthine was detected in significant amounts in any of the biopsy specimens. In addition, uric acid concentrations were consistently below the upper limit of normal plasma concentrations in our institution (2 µmol/g dry weight). Uric acid concentrations were 1.04 ± 0.09 µmol/g before cardioplegic arrest, 1.03 ± 0.07 µmol/g immediately after cardioplegic arrest, and 1.06 ± 0.09 µmol/g after reperfusion.

Figure 3 (left panel) shows the concentrations of creatinine phosphate, the adenylate energy charge, and the levels of HEPs. Creatinine phosphate concentrations decreased from 25.2 to 20.5 µmol/g at 30 minutes of reperfusion but the decrease was not statistically significant. The energy charge decreased significantly from 0.93 to 0.88 during cardioplegia and remained unchanged with reperfusion. The level of HEPs decreased significantly from 4.1 to 31.3 µmol/g at 30 minutes of reperfusion.

Figure 3 (right panel) shows the levels of total adenine nucleotides, total adenine nucleotide degradation products, and total adenine nucleotides and degradation products. Total adenine nucleotide level decreased from 18.0 to 13.7 µmol/g on reperfusion. The level of total adenine nucleotide degradation products increased from 0.43 to 1.31 µmol/g after cardioplegia and then decreased to 0.96 µmol/g upon reperfusion. The level of total adenine nucleotides and precursors decreased 85% from the prearrest values of 18.5 to 15.7 µmol/g 30 minutes after reperfusion.

Table 2 shows the ratios of ADP and AMP to ATP. The ratio of ADP to ATP doubled during cardioplegia from 0.15 to 0.25 and remained unchanged with reperfusion. The ratio of AMP to ATP increased three times from the initial value (0.02) until after cardioplegia (0.05) and was unchanged with reperfusion.

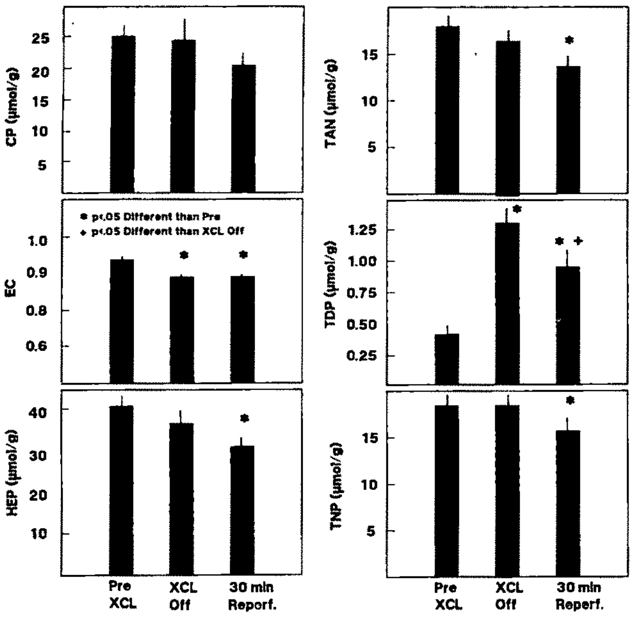

Fig 3. (Left) Concentrations of creatine phosphate (CP), *the adenylate energy charge (calculated by the formula:* EC = *[ATP + 0.5 ADP]/[ATP + ADP + AMP], where ADP = adenosine diphosphate, AMP = adenosine monophosphate, and ATP = adenosine triphosphate), and the high-energy phosphates* (HEP = *ATP + CP). (Right) Total adenine nucleotides* (TAN = *ATP + ADP + AMP), total adenine nucleotide degradation products* (TDP = *adenosine + inosine + hypoxanthine), and total adenine nucleotides and their degradation products* (TNP = *ATP + ADP + AMP + adenosine + inosine + hypoxanthine).*

Comment

Our study suggested two explanations for the postoperative decrease in HEPs. First, the decrease in ATP and the adenylate charge despite an increase in AMP suggested inadequate mitochondrial energy production. Second, the decrease in adenine nucleotide degradation products after reperfusion suggested a washout of diffusable precursors limiting the regeneration of HEPs by salvage pathways.

Measurements

The concentrations of ATP we found before cardioplegic arrest were less than we have previously reported using our spectrophotometric technique [4, 5, 7]. However, the spectrophotometric technique may not be as specific as high-performance liquid chromatography and may have included other metabolic products in addition to ATP. It was difficult to compare our results with the adenine nucleotide concentrations reported by two groups at Duke University [10–12]. Their canine results were reported as nanomoles per gram of protein. Because dried cardiac muscle contains approximately 80% protein, we reduced their results by 80% to permit comparison with our measurements. Their range was 20 to 25 μmol/g dry weight for normal canine left ventricular ATP, 4 to 5 μmol/g dry weight for ADP, and 0.4 to 0.5 μmol/g dry weight for AMP. Our ATP concentrations were 80% and our ADP and AMP concentrations were 60% of their measurements. Patients with coronary artery disease may have lower cardiac adenine nucleotide concentrations than normal dogs. Flameng and colleagues [13] found that the degree of coronary stenosis determined the preoperative adenine nucleotide concentrations. Their concentrations of ATP, ADP, and AMP and total adenine nucleotides in patients with 90% to 100% stenosis were very similar to the concentrations in our study. The concentrations of adenosine, inosine, hypoxanthine, adenylate energy charge, total nucleotide degradation products, and total adenine nucleotides and degradation products we found were similar to those in previous reports [10–13].

Table 2. Mitochondrial Function

Ratios	Before Cardioplegia	After Cardioplegia	After Reperfusion
ADP/ATP	0.15 ± 0.01	0.25 ± 0.01^a	0.24 ± 0.02^a
AMP/ATP	0.02 ± 0.01	0.05 ± 0.01^a	0.05 ± 0.01^a

ADP = adenosine diphosphate (μmol/g dry weight); AMP = adenosine monophosphate (μmol/g dry weight); ATP = adenosine triphosphate (μmol/g dry weight).

[a] $p < 0.05$ versus before cardioplegia.

We found minimal cardiac concentrations of inosine monophosphate, suggesting that the human heart has minimal levels of the enzyme AMP deaminase, which catalyzes the breakdown of AMP to inosine monophosphate. Similar results were found in the canine myocardium [10–13]. In addition, the high levels of hypoxanthine without significant levels of inosine monophosphate suggests that the enzymes hypoxanthine phosphoribosyltransferase and adenylosuccinate synthetase are not used by the human heart for restoration of AMP from adenine nucleotide precursors (Fig 1).

We also showed that xanthine concentrations were minimal in the human heart. The lack of xanthine and the low concentrations of uric acid despite high concentrations of hypoxanthine after reperfusion suggests that xanthine oxidase activity was not significant in the human heart (Fig 1). We found significant xanthine accumulation in the canine heart after ischemia and cardioplegia, suggesting that xanthine oxidase activity is significant in dogs (unpublished observations). Therefore, allopurinol (which inhibits xanthine dehydroxygenase/oxidase activity) may not be effective treatment for humans.

Effects of Cardioplegia

Cold BCP did not prevent the decrease in HEPs despite a threefold increase in AMP. Therefore, intermittent infusions of cold, oxygenated BCP did not permit adequate mitochondrial energy production. The decrease in adenine nucleotides was similar to brief periods of normothermic global ischemia or more prolonged periods of hypothermic global ischemia [11, 12]. The increase in adenine nucleotide degradation products (0.88 μmol/g dry weight) corresponded to the decrease in total adenine nucleotides (3.10 μmol/g dry weight). Therefore, cold BCP did not prevent the degradation of AMP.

Effects of Reperfusion

Reperfusion did not restore mitochondrial energy production because ATP and the adenylate charge did not recover 30 minutes after cross-clamp release. Reperfusion significantly decreased total adenine nucleotide degradation products, suggesting a washout of soluble precursors during reperfusion with warm oxygenated blood, after cold BCP. Sarcolemmal injury during reperfusion may have limited the restoration of HEPs because of inadequate substrates for adenine nucleotide salvage. These results support the concept of a postoperative "reperfusion" injury.

Interventions to Preserve High-Energy Phosphates After Cardioplegic Arrest

The decrease in HEPs may have resulted from inadequate cardioplegic delivery beyond critical stenosis, or inadequate protection despite cardioplegic delivery, or both. Retrograde cardioplegia may improve cardioplegic delivery, and topical ice slush may provide uniform cardiac hypothermia. In a recent study, we found that profound cardiac hypothermia during cold BCP did not preserve HEPs [14]. In addition, we found that glutamate and aspartate administered with a terminal warm BCP infusion did not restore mitochondrial energy production after an elective coronary bypass operation [15]. The infusion of insulin during reperfusion may stimulate postoperative mitochondrial glucose oxidation [16]. Restoration of adenine nucleotide precursors may require a combination of inhibitors of nucleotide and nucleoside degradation and an infusion of nucleotide precursors [9, 17].

Blood cardioplegia provided excellent protection for elective coronary revascularization but resulted in inadequate mitochondrial energy production in the early postoperative period. This defect in myocardial metabolism may have resulted from both mitochondrial dysfunction and a washout of soluble adenine nucleotide precursors.

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Blunt Injuries to the Aortic Arch Vessels

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Thirty patients with 33 vascular injuries from blunt trauma to the brachiocephalic branches of the aortic arch are reported. To our knowledge, this is the largest series reported to date of blunt injuries to these vessels. Mechanisms of injury included deceleration, traction, and crush. Half of the injured vessels were the innominate artery, and a quarter each were the common carotid and subclavian arteries. Common associated injuries were head injuries, hemopneumothorax, lung contusion, long bone fractures, and brachioplexus injuries. Widened mediastinum and extrapleural hematoma were common radiographic findings, and aortic rupture was frequently suspected. Angiography was performed in all patients to identify precisely the nature and site of the injury. Surgical approaches varied with the anatomical site of the injury and required consideration of vascular control in chest, neck, and upper extremity. Twenty-seven patients are alive 6 months to 10 years after injury. Eighteen of 20 vascular reconstructions were patent at follow-up. No patient with brachioplexus injury had return of neurological function.

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There are few reports of extensive experience with great vessel injury from blunt chest trauma [1, 2]. Many patients die on the scene, and for those reaching the hospital, multiple associated injuries including severe neurological injury or aortic rupture may overshadow the vascular trauma [3, 4] and delay diagnosis. The anatomical location of this injury at the interface between neck, chest, and upper extremity complicates surgical exposure and makes precise localization of the injury by arteriography particularly helpful in operative planning. We report the combined experience of two institutions in the management of trauma to the brachiocephalic branches of the aortic arch sustained from blunt trauma.

Patients and Methods

Thirty patients with 33 injuries to the brachiocephalic artery from blunt trauma were admitted to the SUNY Health Science Center at Syracuse, New York, or to the Hartford Hospital, Hartford, Connecticut, between 1978 and 1985 and were managed by one or more of the authors. Twenty-seven patients were admitted with acute injury and 3 had false aneurysms from previous injury. Penetrating injuries to these structures were specifically excluded. The 22 male and 8 female patients ranged in age from 16 to 60 years. In addition to clinical examination in the emergency room, all patients underwent chest radiography and arteriography by standard techniques. Operations were performed under general anesthesia with tracheal intubation.

Injuries of the proximal left and right common carotid arteries were exposed through median sternotomy with cervical extension along the anterior border of the appropriate sternomastoid. Proximal right subclavian injuries were surgically exposed through a median sternotomy with extension to the right supraclavicular fossa. Supraclavicular and infraclavicular cervical exposures were used for access to distal subclavian and proximal axillary arteries. Clavicular resection was added if necessary. The proximal left subclavian and the descending thoracic aorta were exposed through a high left posterolateral thoracotomy. Arterial reconstruction techniques included lateral arteriorrhaphy, primary end-to-end anastomosis, and interposition grafting with either synthetic graft or autogenous saphenous vein.

Results

Mechanisms of Injury

Of 30 patients, 27 were injured in motor vehicle accidents and 3 sustained industrial crush injuries. Distribution of injuries was as follows: innominate artery, 17; subclavian artery, 8; and common carotid artery, 8. Four patients (13%) had aortic rupture concomitant with their brachiocephalic arterial injury (Fig 1). The mechanism of injury was deceleration in 19, traction in 8, and crush in 3.

Diagnosis

Of 27 patients who were seen with acute injury, initial clinical examination revealed decreased extremity pulses in 16, physical evidence of chest wall trauma in 15, and hypotension in 18. Associated injuries were common and included head injuries in 14, long bone fractures in 19, tracheal/bronchial injury in 3, facial injuries in 14, brachial plexus injury in 6, and abdominal trauma in 7.

Chest roentgenograms showed a widened mediastinum or extrapleural hematoma, or both, in 28 of 30 patients (93%). All patients underwent angiography to define the anatomical site of vascular injury. Angio-

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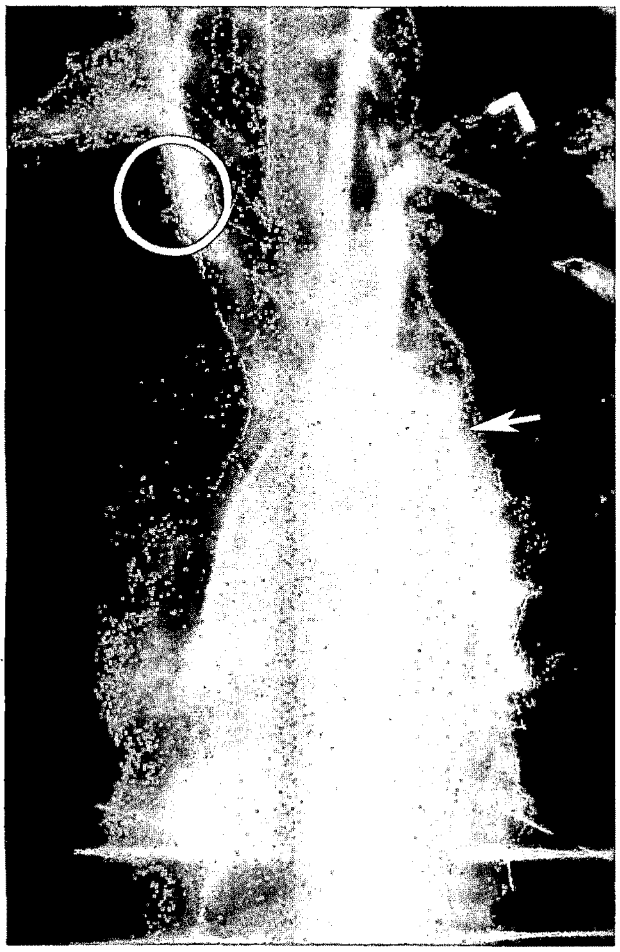

Fig 1. Concomitant aortic (arrow) *and innominate artery* (circle) *rupture.*

graphic evidence of injury included occlusion, false aneurysm, intimal flap, and free hemorrhage (Fig 2).

Management Results

One patient died of her head injury before operative intervention. Two patients died in the operating room during operation; thus, the mortality after arrival at the hospital was 10%. Thirty-day operative mortality was 6.6%.

Eighteen patients underwent a total of 20 arterial reconstructions, 4 patients underwent ligation of a subclavian artery, and 5 patients with intimal injuries were observed and did well without operation.

Twenty-two patients underwent transsternal exploration with or without neck extension for innominate, proximal common carotid, or proximal right subclavian injuries. Ten patients underwent posterolateral thoracotomies for left subclavian injury, with or without concomitant aortic injuries. Five patients were explored with a combined supraclavicular infraclavicular approach for injury to the distal subclavian arteries. (Several patients required more than one approach to accomplish reconstruction.)

Graft Patency

Eighteen of 20 arterial reconstructions were patent at follow-up up to 10 years after operation. Two grafts occluded secondary to graft infection. One of these infections occurred during the initial hospitalization, and one occurred 3 years after injury. Both grafts were removed (one with staged reconstruction) without sequelae. Six patients had brachial plexus injuries, and none of them had return of neurological function. It should be noted that these patients had major skeletal injuries to their upper extremities and had upper extremity amputations either during the initial hospitalization or later.

Comment

Blunt injuries involving major arterial branches of the aortic arch are uncommon; this series appears to be the largest single series reported in the English-language literature to date.

Distribution

The innominate artery is the most frequently injured vessel, followed by the subclavian arteries. Castagna and Nelson [5], in a review of the entire English-language literature in 1975, reported 22 injuries to the innominate artery and 14 to the subclavian artery. Common carotid injuries are infrequently reported, although in the present series they were as frequent as subclavian injuries.

Mechanisms of Injury

In the present series, blunt injuries could be categorized as deceleration, crush injury, or traction on the upper extremity or neck. The exact mechanism through which impact injuries to the aortic arch branches occurs has not been well defined. Binet [6] and other investigators have hypothesized that there is compression to the mediastinal contents between the vertebral column and the sternum in the classic deceleration injury best exemplified by a driver in a motor vehicle crash who strikes the steering wheel with the chest. This sudden compression displaces the heart into the left hemithorax, putting the convex right anterior lateral wall of the aorta under tension. Further tension is placed on the innominate and subclavian arteries if there is hyperextension of the neck or rotation of the head, both of which frequently occur in blunt trauma. Distraction and hyperextension of the shoulder joints are common factors involved with injury to the subclavian artery as well as more distally in the brachial and axillary arteries. The net result of these mechanisms is an acute stretching of all or a portion of the wall of the brachiocephalic arteries, producing intimal tears, disruption of the media, or complete transection of the artery. These wall injuries may in turn lead to dissection, thrombosis, pseudoaneurysm, or frank hemorrhage [7, 8]. In addition, we have observed direct crush injury to both common carotid and subclavian arteries.

Clinical Presentation

Exsanguinating hemorrhage from blunt arterial injuries to aortic arch vessels is uncommon. Signs or symptoms of arterial occlusion are more frequent than massive hemor-

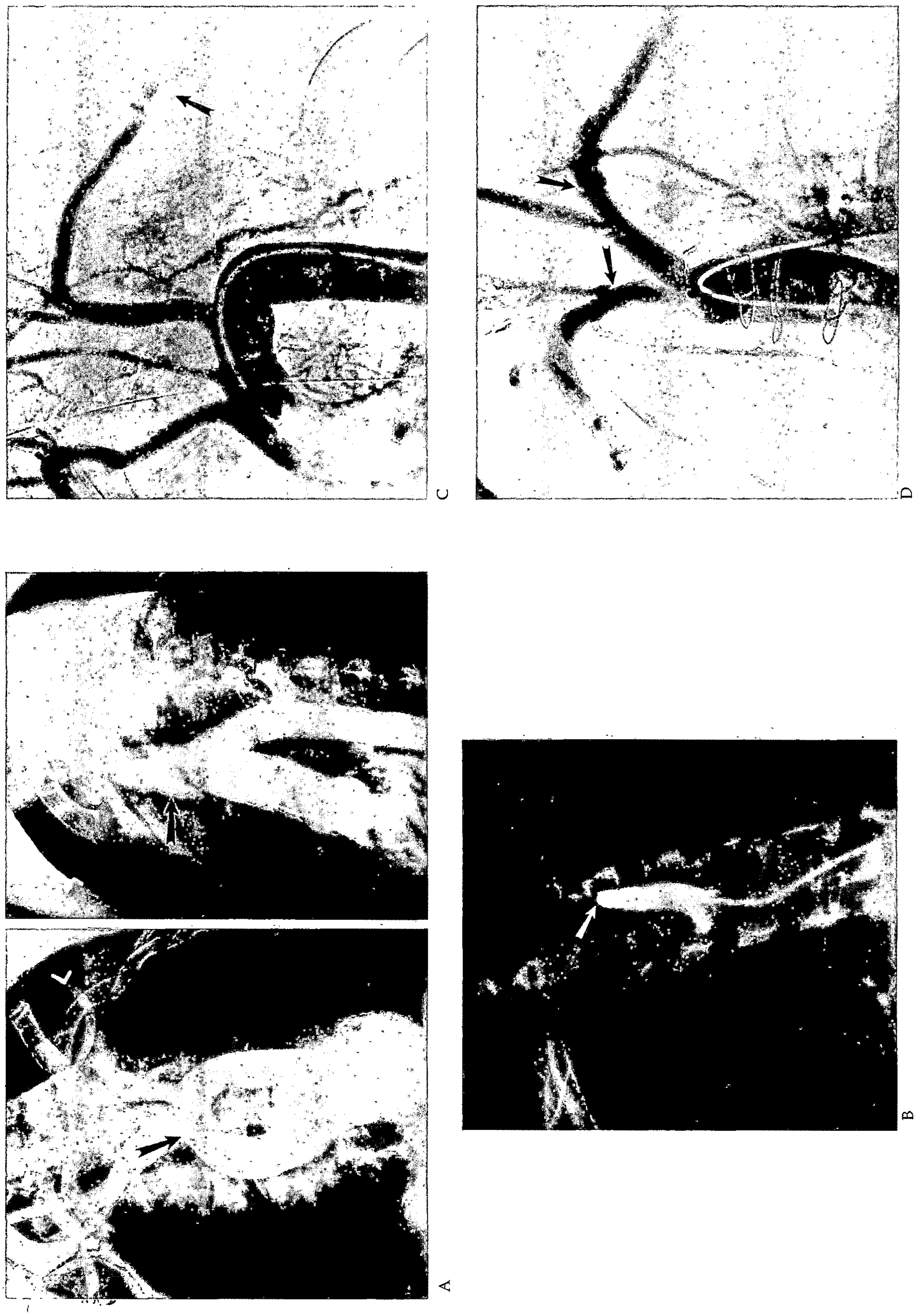

Fig 2. Various forms of presentation of blunt arterial damage: (A) pseudoaneurysm; (B) occlusion; (C) hemorrhage, demonstrated by filling of nonvascular structure; (D) intimal.

rhage. When a full-thickness laceration or transection occurs, the hematoma is often contained within adventitia and periadventitial tissues, arresting hemorrhage. Contained hematomas may lead to late presentation as a false aneurysm many years after injury.

Decreased or absent pulses in the upper extremity, signs of chest wall trauma, and hypotension are the most common findings on initial physical examination (although hypotension may be caused by the aortic arch branch injury, it may also be from an associated injury). Palpable pulsatile mass in the supraclavicular fossa, symptoms of upper extremity ischemia, bruits in the supraclavicular fossa or over the upper chest, superior vena caval syndrome, recurrent nerve palsy, subclavian steal, and dysphagia have all been reported as evidence of acute injury or signs of false aneurysm remote from the injury [8]. Multiple injuries are common in these patients, which may lead to a delay in diagnosis and an increased mortality [3, 9]. Closed head injury and brachial plexus injury are particularly devastating in this setting. The force of injury required to disrupt the aortic arch branch is severe and the associated head injury may be lethal. Traction injuries severe enough to disrupt distal subclavian or proximal brachial arteries may cause permanent dysfunction of the brachial plexus even without anatomical disruption. None of our patients who was seen with acute injury with loss of brachial plexus function had return of neurological function.

Radiographic Findings

A wide mediastinum is often observed on chest roentgenograms [10] and was noted in 93% of our patients. Because of the greater awareness of aortic injury, this may lead to suspicion of descending aortic tear. Four of our patients indeed had a concomitant aortic tear at the ligamentum arteriosum; the incidence of concomitant tear with aortic arch branch injury varies between 4% and 12% [11].

Injury to aortic arch branches with blunt trauma should be suspected in patients with decreased or absent upper extremity or neck pulses, large cervical hematomas, major intrathoracic hemorrhage, brachial plexus injury, or evidence of mediastinal widening on plain chest roentgenogram. We believe that angiography should be performed in all stable patients suspected of having a vascular injury of the aortic arch branches. Arteriography should include visualization of all the brachiocephalic arteries as well as the common and internal carotid arteries. This will allow diagnosis of occult injuries in other brachiocephalic arteries and distinction between brachiocephalic artery injury and thoracic aortic injury or a combination of the two. Furthermore, operative planning is more precise and conduct of operation more efficient if the anatomical site of the injury is precisely identified.

The angiographic evidence of vascular injury that we identified included delayed flow, pseudoaneurysms, intimal flap, occluded artery, free extravasation, and dissection [12]. Although few of our patients underwent computed tomographic scans or magnetic resonance imaging, these methods may be of some use in the future, especially as ultrafast contrast-enhanced computed tomographic scans and cardiac gating magnetic resonance imaging are perfected.

Operative Exposure

Contingency planning must include consideration of exposure within the chest as well as the neck and upper extremity. In the supine position, median sternotomy with neck and upper extremity extension is the most versatile approach, providing exposure of all the bracheocephalic branches except for the proximal left subclavian artery [13]. It does not, however, allow adequate exposure for repair of a concomitant aortic injury at the ligamentum arteriosum. The pericardium is opened to expose the ascending aorta and the innominate artery is exposed at the pericardial reflexion. The proximal left common carotid artery is easily found by dissection further distally along the arch. The innominate vein can be retracted or divided if necessary [14, 15].

Optimal exposure of the proximal left subclavian artery alone is best obtained through a high posterolateral thoracotomy (third or fourth interspace). In the lateral position, extrathoracic control of the distal subclavian artery may be obtained by extending the posterolateral thoracotomy incision cephalad between scapula and spine. This elevates the scapula off the chest wall to expose the first rib, over which passes the subclavian artery. Exposure of the subclavian artery is limited but allows placement of the vascular clamp.

Concomitant aortic injury at the ligamentum arteriosum can be repaired through the same approach. This posterolateral incision can also be used for combined injuries to the esophagus and tracheal/bronchial tree when applicable. In the present series, it was used for injury to the left subclavian artery combined with transection of the left main bronchus. If, however, an aortic injury at the ligamentum arteriosum is present with an injury to one of the anterior brachiocephalic arteries, simultaneous exposure of all injuries may not be possible. The surgeon would be forced to repair them in sequence depending on the relative priority of urgency.

Combined supraclavicular and infraclavicular incisions provide excellent exposure for operative procedures on the distal subclavian and axillary arteries. They may be joined with resection of the medial half of the clavicle if necessary to increase visualization, particularly if there is extensive hematoma to complicate the view. Careful division of the scalenus anticus muscle with special care taken to protect the underlying artery and brachial plexus helps open up the supraclavicular exposure of the midsubclavian artery.

Exposure in the supraclavicular fossa is particularly limited, and direct entry into a hematoma in this location should not be attempted before initial proximal and distal vascular control is achieved.

Once vascular exposure and control is achieved, vascular reconstruction is tailored to the particular injury and includes lateral arteriorrhaphy and anastomosis/reimplantation with grafting using either autologous tissue or prosthetic material.

Hospital mortality for blunt injuries of brachiocephalic arteries have been reported to be between 10% and 35%;

our patient injury mortality was 10% and our operative mortality was 6.6%. Graft patency is good both in the literature and in our series, with a 90% graft patency rate at a mean of 5 years of follow-up. Although two graft occlusions in our series that were secondary to infection were subsequently handled without major problems, potential advantages of use of autologous material in reconstruction are implied.

Although arterial reconstruction is preferable [16, 17], ligation of the subclavian artery can be performed with safety to the limb in the severely unstable patient or in the midst of bacterial contamination. There is excellent collateral flow around the proximal subclavian artery, and limb-threatening ischemia is extremely rare. If disabling ischemic symptoms occur in follow-up, cervical reconstruction can be performed.

Associated venous injuries, of which there were four in our series, should be repaired when feasible. Whether to explore all patients with evidence of arterial injury after blunt trauma is a matter of some concern and controversy. We followed 5 patients with radiographic evidence of intimal injury without sequelae (4 of the patients had fairly minor injury without bruit, without arm blood pressure differences, and without evidence of full-thickness injury). In 1 patient, intimal injury extended all the way up the internal carotid artery to the skull and could not be repaired. Although anatomical abnormality remains, the patient remains asymptomatic and well 4 years later. If the diagnosis is made late after injury and there appears to be only minor vascular trauma without any associated vascular symptoms, we believe that a period of observation is justified, followed by repeat angiography. If 1 week later no symptoms appear and the angiography reveals no progression, close follow-up can be performed without undue risk to the patient.

Cerebral Protection

Use of a temporary shunt for cerebral protection during operation on the innominate or carotid artery is still controversial. Our experience with chronic occlusive disease of the carotid bifurcation as well as the aortic arch branches has demonstrated for us the reliability of selective shunting on the basis of the "stump pressure" measured distal to the clamp on an extracranial artery. This technique for assessing adequacy of collateral flow was originally advocated by Ehrenfeld and associates [18], and we have adopted their initial suggestion of a cutoff pressure of 50 mm Hg. The abundance of collateral flow around the proximal extracranial arteries is such that shunting is rarely necessary. All of our patients had "stump pressures" greater than 50 mm Hg when their innominate or common carotid artery was clamped. None of the patients was shunted, and no patient had a neurological deficit or a worsening of a neurological deficit related to the absence of shunting.

Cardiopulmonary Bypass

In earlier reports of innominate artery repair, cardiopulmonary bypass was used. With a deeply curved tangential occlusive clamp to control the origin of the innominate or common carotid artery, cardiopulmonary bypass is usually not required. Cardiopulmonary bypass is useful in repairing extensive injuries of the brachiocephalic artery that continue on the convex portion of the ascending aorta and transverse arch itself. Extensive injuries that involve both the innominate and left common carotid (which can arise from a common trunk) are particularly hazardous because interruption of both these major blood supplies to the brain requires some sort of shunting or bypass for cerebral protection. We preferred to use cardiopulmonary bypass combined with circulatory arrest for this particular situation. Cardiopulmonary bypass can be useful in returning autologous blood to the patient but has the disadvantage in this setting of requiring systemic heparinization. In contrast, current autotransfusion techniques are more easily performed and do not require heparinization [19, 20]. Cardiopulmonary bypass was required for one of the vascular repairs in this series.

Associated Injury

Associated injuries are common and include nonvascular thoracic injury, head injury, long bone fractures, tracheobronchial tears, and brachial plexus injuries. Head injury is the leading nonvascular cause of death reported in the literature and occurring in our series [13]. Injuries to the subclavian arteries were often associated with brachial plexus injuries in the literature and in our series. We believed that the injuries were mainly secondary to traction, with disruption of nerve roots. These latter injuries are particularly debilitating, and none of our patients have improved neurologically after such injury [21].

Conclusion

We conclude that prompt and precise diagnosis combined with early operation and optimal surgical exposure for control of hemorrhage are important facets in the successful management of these patients. Associated neurological injuries (brain and brachioplexus) are common and may leave lasting deficits. Angiography is essential and allows identification and precise anatomical location of such injuries, which facilitates operative planning.

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Hard-Shell Cardiotomy Reservoir for Reinfusion of Shed Mediastinal Blood

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We conducted a prospective, randomized, controlled trial comparing homologous blood consumption between groups of patients receiving conventional mediastinal drainage (group 1) or reinfusion of shed mediastinal blood (group 2) using a hard-shell cardiotomy reservoir. One hundred consecutive patients who had elective coronary artery or valvular operations were studied. The two groups were comparable with regard to age, sex, weight, preoperative and postoperative hemoglobin levels, and surgical procedure. Group 2 patients had their shed mediastinal blood reinfused for up to 18 hours postoperatively; otherwise, the two groups were treated identically. For groups 1 and 2, average mediastinal blood losses were 705 ± 522 and 822 ± 445 mL and homologous blood consumption was 3.83 ± 2.58 and 3.15 ± 2.05 U, respectively (neither measure was significantly different). However, if blood losses exceeded 500 mL, there was a statistically significant reduction in homologous blood requirements in group 2 as compared with matched controls in group 1. This difference was most significant in patients with the greatest mediastinal losses.

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Reinfusion of shed mediastinal blood after cardiac operations was first reported by Schaff and colleagues [1], who were able to demonstrate a reduction in homologous blood consumption. This finding was confirmed by Johnson and co-workers [2], but Thurer and associates [3] were not able to demonstrate any significant saving of blood when this technique was used as part of a coordinated blood conservation program. These three studies [1–3] and a similar study [4] each used a Sorenson Receptaceal Autotranfusion system (Sorenson Research, Salt Lake City, UT), which has provoked criticism related to its inconvenience of use, cost, and potential for bacterial contamination [5].

Use of a hard-shell cardiotomy reservoir, initially reported by Weniger and colleagues [6], appears to alleviate most of these problems [5]. However, few published data describe the bacterial safety or the ability of a hard-shell cardiotomy reservoir to reduce homologous blood requirements when used in this manner. Any conservation study will inevitably improve general awareness of the need for conservation. We are unaware of any prospective controlled study that examines the efficacy of a hard-shell cardiotomy reservoir when used for reinfusion of shed mediastinal blood.

We undertook a prospective controlled study to investigate whether a hard-shell cardiotomy reservoir does contribute to a saving in homologous blood consumption as compared with conventional drainage and whether it is bacteriologically safe when used for this purpose.

Material and Methods

A consecutive series of 100 patients hospitalized at the Regional Adult Cardiothoracic Unit, Broadgreen Hospital, for an open heart operation were randomly allocated to receive either conventional postoperative mediastinal drainage (group 1) or reinfusion of shed mediastinal blood (group 2). One male patient in group 1 was excluded from the analysis of results because of postoperative gastrointestinal bleeding.

Patients who underwent coronary bypass grafting received dipyridamole 100 mg every eight hours for 48 hours before operation. Those patients who received anticoagulation therapy had administration of warfarin stopped 48 hours preoperatively.

Perioperative and postoperative management was standardized. Patients with a hematocrit greater than 30% had a unit of blood withdrawn immediately before cardiopulmonary bypass; this was reinfused in the postbypass period. A perioperative anemia of 17% was accepted; postoperatively, homologous blood was not transfused unless the hematocrit was less than 30%. Anticoagulation was established with heparin to achieve an activated clotting time (ACT) greater than 480 seconds; this level was maintained throughout the period of cardiopulmonary bypass with additional heparin as indicated by repeated estimations of the ACT. At the end of bypass, anticoagulation was reversed with protamine sulfate at a ratio of 1 mg of protamine to 100 U of existing heparin as judged by the last ACT measurement.

In group 1 patients a Polystan soft-shell cardiotomy reservoir (Polystan A/S Walgerholm 8, DK-2750 Ballerup, Denmark) was used during bypass, and in group 2 patients a Bentley Catr hard-shell cardiotomy reservoir (Bentley-Edwards CVS Division, Baxter Healthcare, New-

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bury, England) was used. A variety of both membrane and bubble oxygenators were used in the following distribution: group 1, 72.5% Cobe CML (Cobe Laboratories, Lakewood, CO) (membrane), 23.5% Polystan Rygg (bubble), and 4% Bentley Ben10 plus (bubble); group 2, 70.8% Cobe CML, 27.1% Polystan Rygg, and 2.1% Bentley Ben10 plus.

After bypass, any residual blood left in the perfusion circuit was saved and infused through a peripheral vein. Both groups of patients had pericardial and mediastinal sump drains (Axiom Medical, Paramount, CA). Blood from group 1 was drained into conventional drainage bottles with an applied suction of 25 cm H_2O. In group 2 patients, both drains were connected to the top of the cardiotomy reservoir, previously used during bypass, and a suction of 50 cm H_2O was applied.

Group 2 patients had their shed mediastinal blood returned to them. Blood loss was initially measured every 15 minutes, and this volume was reinfused into the patient over the next 15-minute period with an IMED (Oxford, England) volumetric pump; a buffer of 50 mL of blood was always maintained in the cardiotomy reservoir. As the rate of bleeding decreased, the periods of observation and reinfusion were extended to 30 minutes and finally to hourly periods. In all other respects, both groups were managed in the same way. Homologous blood or hetastarch was infused to maintain cardiovascular stability and a hematocrit of 30%. Fresh-frozen plasma or platelets were administered on the basis of continued bleeding if the coagulation profile was abnormal.

Reinfusion of shed mediastinal blood was continued up to a maximum of 18 hours. At this time, a sample of blood was taken from the cardiotomy reservoir for bacteriology culture. Thereafter, the cardiotomy reservoir was used as a simple reservoir, without further reinfusion.

Hemoglobin levels were compared preoperatively and 1 week postoperatively. Data for consumption of homologous blood during the entire hospitalization were obtained from computerized laboratory records. Data were analyzed with paired and unpaired Student's t test; a p less than 0.05 was considered significant.

Results

The demographic data of the two groups are shown in Table 1, and the details of the operative procedures undertaken are shown in Table 2. There were no significant differences between the two groups. The mean mediastinal drainage for group 1 was 705 ± 522 mL, not significantly different from the drainage in group 2 (822 ± 445 mL). Group 1 patients required a mean of 3.83 ± 2.58 U of homologous blood, whereas group 2 patients required 3.15 ± 2.05 U of homologous blood; this difference did not achieve statistical significance. Six patients in each group did not require homologous blood transfusion.

When patients within the groups were stratified on the basis of mediastinal blood loss, there was a statistically significant difference in blood consumption between the two groups that occurred when mediastinal blood loss was more than 500 mL (Fig 1).

Fresh-frozen plasma was administered to 10 patients in group 1 and to 11 patients in group 2. One patient in each group received a platelet transfusion. Within each group, there was a marked decrease in platelet count in the postoperative period, and a pronounced return toward the normal range for the prothrombin ratio. However, there was no significant difference between the groups (Table 3).

One patient in group 2 required reexploration for surgical bleeding and received 1,580 mL of reinfused blood but no homologous blood. The greatest volume of blood reinfused was 2,280 mL into a patient with a preexisting coagulopathy (this was achieved without any apparent ill effects). No bacterial growth was evident in the cultures taken at the end of the 18-hour reinfusion period, and no patient developed any blood-borne infection.

Comment

Undoubtedly, despite stringent screening of donated blood, there is an ever-present risk of transmission of infections, particularly viral infections [7–9]. In Britain, where the reported transmission of non-A, non-B hepatitis in donated blood is relatively low after cardiac operations [10], this level of infection will inevitably increase the morbidity and mortality of an otherwise relatively safe procedure [11]. Equally, homologous blood is a finite and expensive resource that should be conserved whenever possible [12]. Therefore, any maneuver that is demonstrably safe should be used to conserve the patient's own blood during a surgical procedure. Cardiac surgery, with

Table 1. Demographic Data

Variable	Group 1 (n = 51)	Group 2 (n = 48)
Age (yr)	56.9 ± 9.4	58.3 ± 8.9
Weight (kg)	77.3 ± 12.1	75.5 ± 12.1
Male:Female	38:14	38:11
Bypass time (min)	126 ± 38	123 ± 28
Hemoglobin (g/dL)		
Preoperative	14.6 ± 1.4	14.6 ± 1.3
Postoperative	10.8 ± 1.4	10.6 ± 1.2

Comparison of demographic data, bypass times, and hemoglobin levels between the two groups. Data are mean ± standard deviation; there are no significant differences between group 1 and group 2.

Table 2. Details of Operative Procedure

Procedure	Group 1	Group 2
Coronary artery bypass grafts	42[a]	41[a]
CABG + one valve	4	3
CABG + two valves	1	. . .
Valve replacement	4[a]	4

Distribution of surgical procedure between the two groups. [a] One reoperation.

CABG = coronary artery bypass graft.

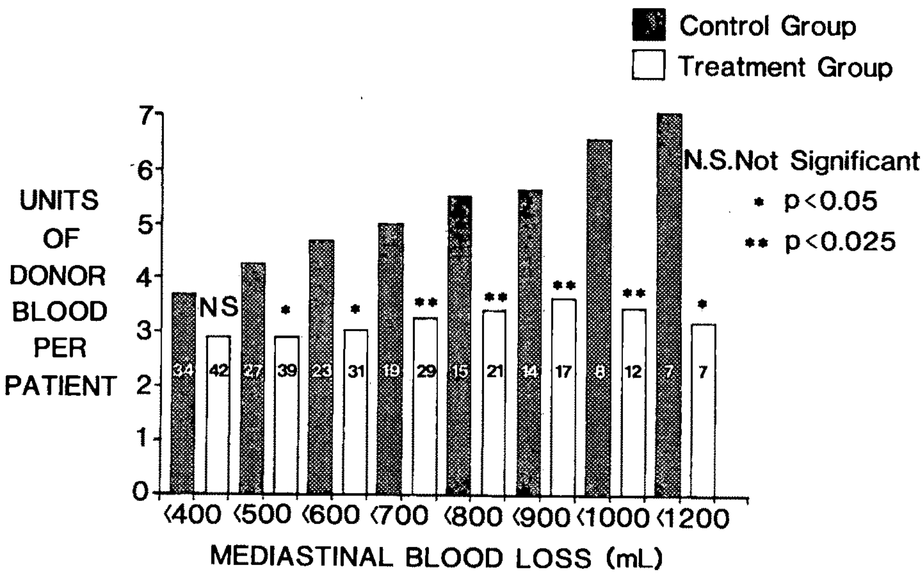

Fig 1. Comparison of homologous blood consumption between the two groups with patients ranked according to their mediastinal blood loss. Numbers within each bar refer to the number of individuals within each rank.

its historically large consumption of homologous blood [13], is an obvious target for conservation measures.

Autotransfusion is one measure that can be easily and cheaply undertaken. This study demonstrates that reinfusion of shed mediastinal blood using a hard-shell cardiotomy reservoir results in a reduction in the use of homologous blood. This saving becomes more marked as the blood loss in the postoperative period increases. The difference in homologous blood consumption between the two groups achieves statistical significance in patients bleeding more than 500 mL. This represents 81% of patients in group 2. The average blood loss for our patients was in the range of 700 to 800 mL, which compares favorably with recent reports from other centers [14–16]. The greater the mediastinal blood loss, the greater the anticipated benefit of reinfusion of shed mediastinal blood. Patients receiving the greatest volume of reinfused blood required no more homologous blood or blood products than those patients who had more modest infusions. In addition, there were no significant differences between the two groups in their measured coagulation profiles during the period of reinfusion, demonstrating that reinfusion of shed mediastinal blood in clinically acceptable amounts does not cause a coagulopathy.

Table 3. Prothrombin Ratios and Platelet Counts at End of Operation and at 18 Hours After Operation

Factor	Group 1	Group 2
Prothrombin ratio		
End of operation	1.57 ± 0.31	1.46 ± 0.27
18 Hours after operation	1.34 ± 0.26	1.31 ± 0.21
Platelet count (× 10^9/L)		
End of operation	151 ± 54	158 ± 56
18 Hours after operation	123 ± 45	136 ± 43

Data expressed as mean ± standard deviation; there are no significant differences between group 1 and group 2.

The cardiotomy reservoir is used in a conventional manner during cardiopulmonary bypass before its conversion for reinfusion. During bypass, it is exposed to a large quantity of air mixed with blood and is thus exposed to the potential of air-borne bacterial contamination. Despite this potential problem, no blood taken from the reservoirs at the end of reinfusion produced any bacterial growth, and no patient showed any evidence of blood-borne infection. On this basis we believe that is is safe to reinfuse shed mediastinal blood for up to 18 hours after cardiopulmonary bypass.

As shown in Fig 1, this study demonstrates that if shed mediastinal blood is reinfused homologous blood requirements become independent of postoperative mediastinal losses. Reinfusion of shed mediastinal blood can only conserve blood lost through mediastinal drains. The observation that group 2 patients still required 3 U of homologous blood per patient clearly indicates that reinfusion of shed mediastinal blood is only one facet of a blood conservation program. Only when all possible methods of conservation are used may cardiac operations be performed without the use of homologous blood. Our average of 3 U of blood per patient, although it compares favorably with those of other British cardiac units [17], reflects the need for a continued and thorough approach to blood conservation to come closer to the ideal of cardiac operation without use of homologous blood.

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Coronary Flow Velocity Waveforms in Aortic Stenosis and the Effects of Valve Replacement

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In 6 patients with pure aortic stenosis, the flow velocity waveforms in the left anterior descending coronary artery were studied using an 80-channel 20-MHz pulsed Doppler velocimeter before and immediately after aortic valve replacement. All patients showed normal coronary arteriograms. The left anterior descending coronary artery flow velocity waveform in aortic stenosis was characterized by a reverse flow in the first half of systole and a slowly increasing diastolic inflow. After aortic valve replacement, the reverse flow in the first half of systole disappeared in all patients, but an end-systolic reverse flow was discerned in 5 of 6 patients. The increasing rate of the diastolic inflow was augmented in all patients. After aortic valve replacement, the time from onset of diastole to the diastolic peak velocity was shortened from 176.8 ± 28.8 to 90.5 ± 18.8 ms ($p < 0.01$), and the diastolic peak velocity increased from 90.5 ± 28.0 to 122.5 ± 17.2 cm/s ($p < 0.05$). Blood pressure and heart rate, however, did not change significantly before and after valve replacement. These changes in the left coronary artery velocity waveforms after valve replacement suggest the beneficial effects of removal of aortic stenosis on human coronary artery inflow.

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Patients with aortic stenosis often demonstrate clinical and electrocardiographic evidence of myocardial ischemia during hemodynamic stress. Several clinical studies have indicated that the causative factors responsible for the development of myocardial ischemia in aortic stenosis are a decreased coronary reserve [1–4] and an oxygen supply/demand discrepancy [2, 5]. Subendocardial underperfusion results in myocardial anaerobic metabolism [2, 6]. Hemodynamic studies of acute aortic stenosis in animals, on the other hand, have demonstrated a decreased systolic flow [7–9], although the net coronary flow per unit myocardial mass did not differ from that under control conditions. The reduced systolic flow may lead to an autoregulatory compensation by increasing diastolic flow. However, detailed measurement of systolic and diastolic coronary artery flow waveforms in patients with aortic stenosis have been largely hampered by methodological limitations. In addition, the change in velocity waveforms after aortic valve replacement in aortic stenosis has not been clarified in human beings or animals.

We analyzed phasic coronary flow velocity waveforms in detail using an 80-channel 20-MHz pulsed Doppler velocimeter without isolation during cardiac operations [10–12]. The purposes of this study were (1) to clarify the characteristics of the coronary artery velocity waveforms in patients with pure aortic stenosis and (2) to evaluate the effects of aortic valve replacement on the phasic configuration of coronary artery velocity waveforms.

Material and Methods

Six patients, 4 men and 2 women with a mean age of 59.8 years who had chronic pure aortic stenosis and who were admitted to Kawasaki Medical School Hospital between January 1986 and September 1988, were studied during cardiac operations before and after aortic valve replacement with a St. Jude Medical prosthetic valve. A cardiac catheterization study was performed before operation, and absence of coronary artery disease was confirmed by coronary arteriography in all patients. The ratio of end-diastolic diameter to doubled posterior wall thickness (R/Th) was measured by ultrasound echocardiography as an index of left ventricular hypertrophy. Three of 6 patients had a congenital bicuspid aortic valve with calcification. Before cardiopulmonary bypass, the waveforms of the volume flow rate in the ascending aorta were measured by an electromagnetic flowmeter (Gould P-2204; Gould Inc, Oxnard, CA). The pressure waveforms in the ascending aorta and left ventricle were measured by direct needle puncture. Blood velocity measurements in segment 7 or 8 (American Heart Association classification) of the left anterior descending coronary artery (LAD) were performed by an 80-channel 20-MHz pulsed Doppler velocimeter developed in our laboratory. The system detects Doppler signals from 80 channels by a zero-cross method and analyzes Doppler signals from one optional channel by a fast Fourier transform method, both in real time [10–12]. The sample volume of our system is $\pi \times 0.5^2 \times 0.2$ mm^2. A specially designed holder with a finger grip [11] was used to fix the transducer at a 60-degree angle on the LAD without dissection of the vessels. The operator followed cardiac motion carefully with his fingers. When 30 to 45 minutes had elapsed after discontinuation of the

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Table 1. Patient Profiles and Hemodynamic Variables Before and After Aortic Valve Replacement

Patient No.	Sex	Age (yr)	Blood Pressure (mm Hg) Before	Blood Pressure (mm Hg) After	Heart Rate Before	Heart Rate After	Ao-LV Pressure Gradient	R/TH	Remarks
1	F	52	110/70	120/60	95	122	100	2.00	Bicuspid
2	M	59	100/70	120/75	105	112	50	1.63	. . .
3	M	68	120/85	95/65	100	114	112	1.81	. . .
4	F	58	90/50	100/60	80	84	110	1.23	Bicuspid
5	M	67	95/65	80/50	125	110	116	1.62	Bicuspid
6	M	57	80/56	100/70	120	130	68	2.58	. . .
Mean ± SD			99.2 ± 14.3/66 ± 12.2	102.5 ± 15.4/63.3 ± 8.8	104.2 ± 16.6	112 ± 15.6	92.7 ± 27.7	1.81 ± 0.46	

Blood pressure and heart rate were measured during the cardiac operation.

Ao-LV pressure gradient = peak systolic pressure gradient between the ascending aorta and the left ventricle obtained at cardiac catheterization study; Bicuspid = congenital bicuspid aortic valve; R/TH = ratio of end-diastolic short-axis diameter of the ventricle to doubled value of posterior wall thickness measured by ultrasound echocardiography; SD = standard deviation.

cardiopulmonary bypass after aortic valve replacement and when a steady hemodynamic state had been achieved, measurements of the flow velocity in the LAD and of the other hemodynamic variables described were repeated. These data and electrocardiographic data were recorded. The profile and hemodynamic data of the patients are shown in Table 1. The mean aorta–left ventricular systolic pressure gradient was 92.7 ± 27.2 mm Hg (range, 50 to 116 mm Hg), and the mean R/Th was 1.81 ± 0.45 (range, 1.23 to 2.58). Blood pressure and heart rate did not change significantly after valve replacement.

The systolic reverse peak flow velocity (V_{SR}), diastolic forward peak flow velocity (V_{DF}), and time from the onset of diastole to the diastolic peak velocity (T_{DPV}) were measured from the LAD velocity waveforms obtained by the envelope of the fast Fourier transform display before and after aortic valve replacement (Fig 1). The ratio of the systolic peak reverse flow velocity to the diastolic peak forward flow velocity (V_{SR}/V_{DF}) was calculated. As a control study, LAD flow velocities were measured in 10 adult patients with atrial septal defect after closure of the defect (mean age, 44.0 ± 5.6 years; 8 men and 2 women), and T_{DPV} and V_{DF} were calculated to compare with those in aortic stenosis.

Data were expressed as the mean ± the standard deviation. Statistical significance was tested by paired and nonpaired t tests. The criterion for statistical significance was a p value less than 0.05.

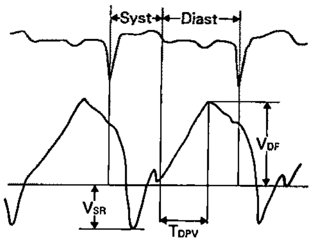

Fig 1. Schematic illustration of three variables extracted from the envelope of the fast Fourier transform display of the phasic blood velocity waveform in the left anterior descending coronary artery. (Diast = diastole; Syst = systole; T_{DPV} = time from onset of diastole to diastolic peak velocity in milliseconds; V_{DF} = diastolic forward peak velocity in centimeters per second; V_{SR} = systolic reverse peak velocity in centimeters per second.)

Results

Figure 2 shows a representative tracing of the LAD velocity waveform in a patient with aortic stenosis (patient 1) before (top) and after (bottom) aortic valve replacement. The characteristics of the flow velocity waveforms are shown in Table 2. The velocity waveforms were characterized by a decreased systolic flow component with a first-half systolic reverse flow and a slowly increasing rate of early diastolic inflow before aortic valve replacement. After aortic valve replacement, the first-half systolic reverse flow disappeared in all patients, but an end-systolic reverse flow appeared in 5 of 6 patients. There was a significantly high correlation coefficient (r = 0.94, $p < 0.01$) between V_{SR}/V_{DF} and R/Th before aortic valve replacement. The value of V_{SR}/V_{DF} decreased from

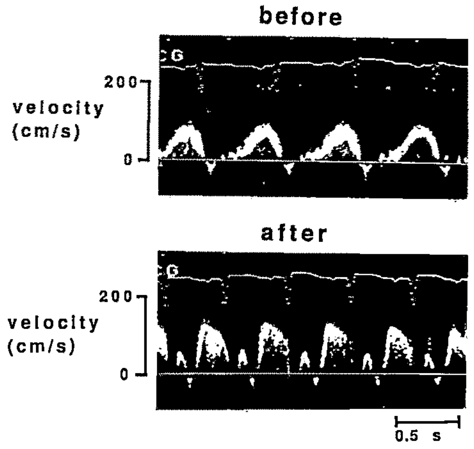

Fig 2. (Patient 1.) A representative left anterior descending coronary artery velocity waveform in a patient with aortic stenosis before aortic valve replacement (top) and after valve replacement (bottom). The velocity waveforms are shown by the fast Fourier transform display (described in detail in text).

Table 2. Changes in Left Anterior Descending Coronary Artery Velocity Waveforms Before and After Aortic Valve Replacement

	First-half Systolic Reverse Flow		End-Systolic Reverse Flow		V_{SR}/V_{DF}		T_{DPV} (ms)		V_{DF} (cm/s)	
Patient No.	Before	After	Before	After	Before	After	Before	After	Before	After
1	+	–	–	+	0.46	0.2	185	92	93	109
2	+	–	–	+	0.53	0.15	154	108	61	112
3	+	–	–	–	0.54	0	169	92	123	137
4	+	–	–	+	0.58	0.05	215	112	123	145
5	+	–	–	+	0.50	0.23	200	77	82	102
6	+	–	–	+	0.4	0.17	138	62	61	130
Mean ± SD					0.50 ± 0.06	0.13 ± 0.10[a]	176.8 ± 28.8	90.5 ± 18.8[a]	90.5 ± 28.0	122.5 ± 17.2[b]

[a] $p < 0.01$ versus before valve replacement; [b] $p < 0.05$ versus before valve replacement.

SD = standard deviation; T_{DPV} = time from onset of diastole to diastolic peak velocity; V_{DF} = diastolic forward peak velocity; V_{SR} = systolic reverse peak velocity.

0.50 ± 0.06 to 0.13 ± 0.10 after aortic valve replacement. The T_{DPV} was 176.8 ± 28.8 ms before valve replacement and 90.5 ± 18.8 ms after valve replacement ($p < 0.01$.) The value of T_{DPV} showed no statistically significant correlation with heart rate either preoperatively or postoperatively. The T_{DPV} in the control group was 64.1 ± 14.1 ms, which was significantly shorter than the values before ($p < 0.001$) and after ($p < 0.001$) valve replacement. The V_{DF} was 90.5 ± 28.0 cm/s before valve replacement and 122.5 ± 17.2 cm/s after valve replacement ($p < 0.05$). The V_{DF} in the control group was 54.56 ± 14.55 cm/s, which was lower than the V_{DF} before valve replacement ($p < 0.001$). These changes in flow velocity values before and after valve replacement are shown in Fig 3. The correlation coefficient between the aorta–left ventricular pressure gradient and T_{DPV} was relatively high ($r = 0.78$) but not statistically significant.

To indicate the temporal location of the systolic reverse flows, representative tracings of aortic and left ventricular pressure curves along with coronary velocity waveforms in the same patient before and after aortic valve replacement are shown in Figure 4. The timing of the systolic reverse flow before aortic valve replacement coincided well with that of the maximal aorta–left ventricular pressure gradient, whereas the peak systolic reverse flow after valve replacement occurred at the dicrotic notch of the aortic pressure waveform. Figure 5 shows a typical tracing of coronary artery velocity waveforms recorded simultaneously with the waveform of the stroke-volume flow rate before and after valve replacement. The timing of the systolic reverse flow before aortic valve replacement occurred just after onset of the aortic stroke–volume flow rate, whereas the systolic reverse flow after valve replacement occurred just after the endpoint of the stroke volume flow rate, ie, at the time at which a small regurgitant flow was observed.

Comment

The major contributions of this study are (1) clarification of the phasic characteristics of the left coronary blood flow in human aortic stenosis and (2) evaluation of the effects

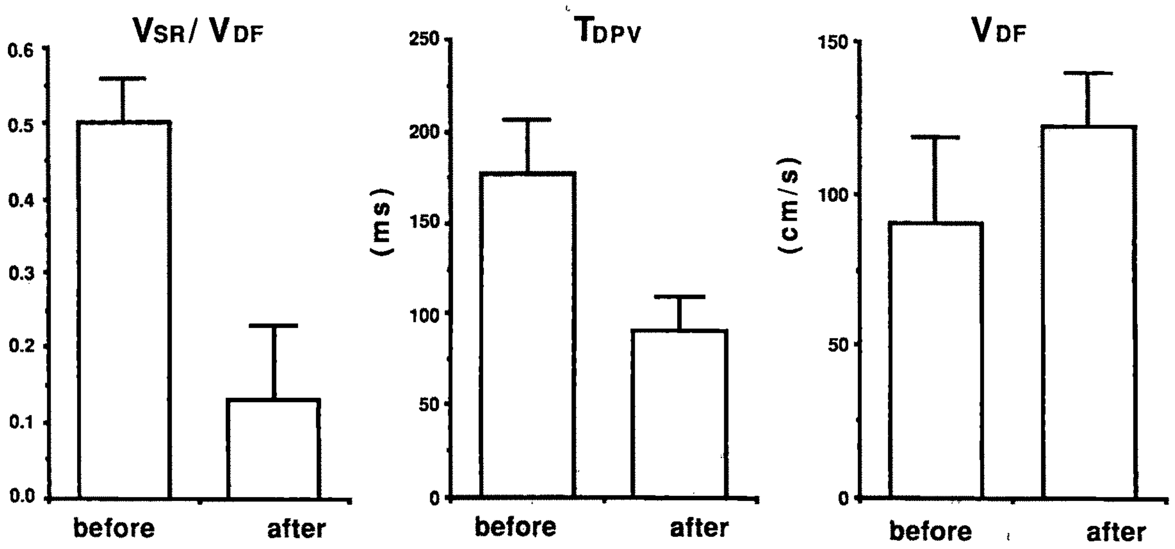

Fig 3. Changes in flow velocity values after aortic valve replacement. (T_{DPV} = time from onset of diastole to diastolic peak velocity; V_{DF} = diastolic forward peak velocity; V_{SR} = systolic reverse peak velocity.)

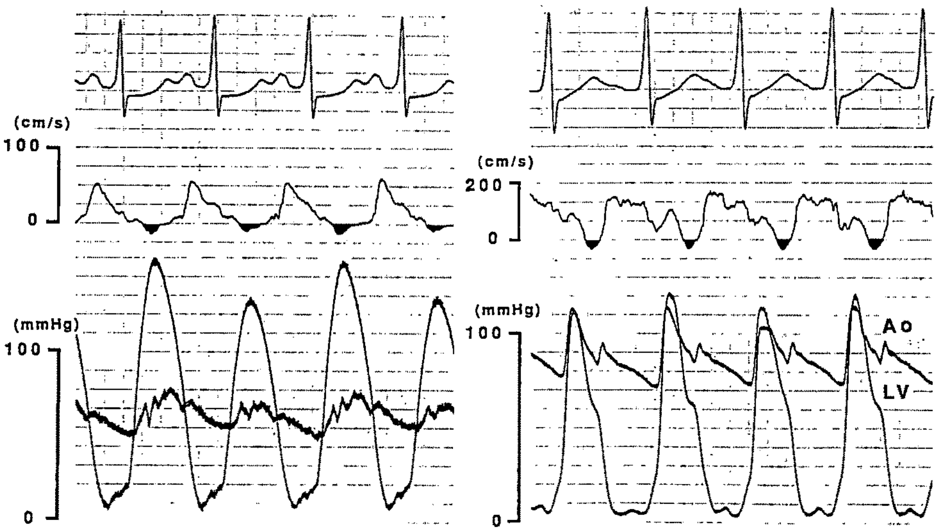

Fig 4. (Patient 6.) A typical example of the temporal relationship between systolic reverse flow (dark shading) *and pressure waveforms in the ascending aorta* (Ao) *and the left ventricle* (LV) *before (left) and after (right) aortic valve replacement. The timing of the systolic reverse flow before replacement coincides well with that of maximal aorta–left ventricular pressure gradient and the reverse flow after replacement occurs at the dicrotic notch of the aortic pressure waveform.*

of aortic valve replacement on coronary artery velocity waveforms. We demonstrated the presence of a reverse flow in the first half of systole and a slowly increasing rate of diastolic coronary inflow and low peak diastolic flow velocity before aortic valve replacement. After aortic valve replacement, the reverse flow during the first half of systole disappeared in all patients, but an end-systolic reverse flow appeared in 5 of 6 patients. In addition, the increasing rate of diastolic inflow was augmented, and the diastolic peak velocity increased.

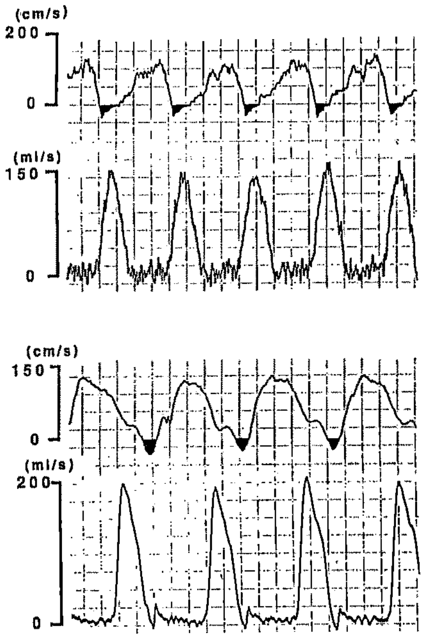

Fig 5. (Patient 4.) A typical tracing showing the temporal relationship between systolic reverse flow (dark shading) *and the aortic stroke–volume flow rate before (top) and after (bottom) aortic valve replacement. Systolic reverse flow before replacement occurred just after onset of the aortic stroke–volume flow rate; after replacement, it occurred just after the endpoint of the stroke–volume flow rate.*

As previously described [10], our velocimeter is equipped with two modes of fast Fourier transform: one is a "fixed mode" in which the fast Fourier transform is performed from a fixed sampling point selected from among 80 channels in advance, and the other is an "automatic mode" in which the sampling point changes automatically among sampling points across the vessel to follow the maximal velocity (the maximal negative flow when the flow is reversed). We measured velocity waveforms by the automatic mode to evaluate the maximal forward (or reverse) flow velocity across the vessel.

As for the systolic reverse flow, Carroll and Falsetti [13] observed it angiographically in the proximal left coronary artery in all their patients with aortic stenosis. In contrast to our results, they observed a small reverse flow in end-systole and interpreted it as an increase in intramyocardial stress. The systolic reverse flow in our observation occurred around the point at which the aorta–left ventricular pressure gradient was maximal. The difference in the timing of the systolic reverse flow may be mainly attributable to the relatively poor time resolution of angiography, because the temporal resolution of our method (2.56 ms) is high. Marcus and colleagues [4] reported that the phasic character of coronary blood flow velocity in patients with aortic stenosis was similar to that in normal subjects, and there was no evidence of systolic reverse flow in aortic stenosis. They may not have detected the systolic reverse flow because the fixed mode zero-cross method they used for analysis of Doppler signals may not be able to detect the peak negative flow across the vessel. After aortic valve replacement, the timing of the systolic reverse flow moved to end-systole, which coincided with a dicrotic notch in the aortic pressure wave and the endpoint of the stroke volume flow rate. These findings suggest that the mechanism of the systolic reverse flow is different before and after valve replacement: the first-half systolic reverse flow may be caused by the force difference in the intramyocardial and aortic pressures, and the end-systolic reverse flow may be caused by the blood discharge from coronary capacitance vessels owing to rapid decrease in the aortic pressure.

Although the fundamental characteristics of blood flow velocity waveforms in the left coronary artery with aortic stenosis were compatible with those of normal subjects (ie, diastolic predominant), we observed a substantial difference in diastolic velocity waveforms. The slowly increasing rate in diastolic inflow velocity was one of the most remarkable velocity waveform findings. It may be caused by increased resistance due to augmented systolic compression of the intramyocardial resistance vessels or decreased filling rate of blood into the intramyocardial vessels due to impaired myocardial relaxation, or both. This speculation may be supported by the high correlation between the time to peak velocity from onset of diastole and the aorta–left ventricular pressure gradient, although it was not significant statistically. Pathophysiologically, the delayed peak velocity in diastole may cause a decrease in diastolic coronary flow and subendocardial ischemia when the heart rate or intramyocardial pressure increases.

The time from onset of diastole to the diastolic peak velocity was shortened significantly (51.1%) after valve replacement, but it was still longer than the normal value measured in 10 patients with an atrial septal defect after closure of the defect. This time delay after valve replacement may be attributable partly to remaining myocardial hypertrophy, as the aorta–left ventricular pressure gradient was almost normalized after valve replacement. Earlier studies have indicated that patients with aortic stenosis usually have a marked increase in net coronary blood flow [3, 14], although the coronary blood flow per unit mass is within the normal range [14]. The diastolic peak velocity before valve replacement was greater than that in the patients with an atrial septal defect, supporting the results of earlier studies. The coronary blood flow velocity further increased by 135%, with disappearance of the first-half systolic reverse flow after valve replacement. This may imply that the decrease in intramyocardial vascular compression force occurred immediately after valve replacement, resulting in the change in magnitude of coronary flow velocity and velocity waveform. The decrease in hematocrit owing to hemodilution by cardiopulmonary bypass may be attributable as well to the higher blood velocity after valve replacement. Other contributory factors such as perfusion pressure and heart rate may not have a significant influence on the change in the blood velocity waveform, because they did not change significantly after valve replacement.

In conclusion, we clarified the detailed characteristics of coronary flow velocity waveforms in human aortic stenosis and the effects of valve replacement on coronary flow dynamics.

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Simultaneous Use of Microfibrillar Collagen Hemostat and Blood Saving Devices in a Canine Kidney Perfusion Model

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Intraoperative autotransfusion and topical microfibrillar collagen hemostats have been increasingly used, often simultaneously, in various surgical procedures to minimize intraoperative blood loss and thus reduce the inherent risks of homologous blood transfusion. As moderate amounts of small particles have been shown to pass through filtering devices during intraoperative autotransfusion, concern has been raised over the amount of heterologous collagen fibrils transfused and their effect on the host. We found that 2% of microfibrillar collagen hemostat particles pass through the 20-μm millipore filter contained in the tested autotransfusion device (William Harvey H-4700 cardiotomy reservoir). Using a canine kidney perfusion model, we found multifocal perivascular inflammatory reactions within the renal parenchyma five days after transfusion of filtered autologous blood containing minute amounts of microfibrillar collagen hemostat. The findings demonstrate a strong inflammatory foreign body response to heterologous collagen particles trapped in the microcirculation of the perfused kidneys. It is concluded that despite using filters with a pore size of 20 μm, using intraoperative autotransfusion and microfibrillar collagen hemostats simultaneously creates a potential risk because adverse reactions can be elicited especially within the microvasculature of tissues containing end-arterial circulation.

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Intraoperative autotransfusion (IAT) has become an established technique in various operative procedures. Many surgical centers routinely transfuse autologous blood during cardiac, vascular, orthopedic, organ transplantation, and emergency room procedures [1–4]. In cardiac operations, filtering devices such as cardiotomy reservoirs are used to collect and return shed autologous blood to the venous circulation intraoperatively [5]. Among the inherent risks of IAT, the potential of transfusing microaggregates and thereby eliciting thromboembolic complications has been addressed [1, 4, 6]. Several studies have evaluated the filtering capacity of autotransfusion units. It has been shown that microaggregates [7–9], bacteria [10], tumor cells [11], bone particles, and debris [12] can pass through filtering devices. Recently, questions have been raised concerning the simultaneous use of IAT and topical microfibrillar hemostats [13]. Microfibrillar collagen hemostats (MCHs) consist of purified fibrillar animal corium collagen. On contact with bleeding surfaces, MCHs effectively reduce surgical hemorrhage by triggering platelet-induced coagulation [14]. Presently, MCHs are widely used to control bleeding during cardiovascular procedures [15–17]. However, simultaneously using IAT for blood salvage and MCHs as a topical hemostat creates the potential of returning blood contaminated with heterologous collagen particles. This study was undertaken to evaluate this risk using an in vivo experimental model.

Material and Methods

The experimental design was approved by the Institutional Animal Care and Use Committee of the University of Pennsylvania, approval No. 860420.

Autotransfusion System

A filtered cardiotomy reservoir (William Harvey H-4700 Biothyl-coated filtered cardiotomy reservoir; C.R. Bard Inc, Billerica, MA) was used in the experiments. The filter consists of a polyester microaggregate screen with a 20-μm pore size, coated with a specifically formulated heparin complex (benzalkonium chloride–heparin complex). Autologous shed blood was collected as described in the following section, mixed with MCHs (Avitene, nonwoven web form; Alcon Inc, Humacao, Puerto Rico), and aspirated into the filtered cardiotomy reservoir using a roller pump with a negative pressure of 20 kP (152 mm Hg). The filtered blood was then transfused via an intraarterial catheter into the renal vasculature. To increase the blood flow to the animal in the gravity system, a 50-mL syringe and a three-way stopcock (Medex Inc, Hilliard, OH) was inserted into the transfusion line, and blood was alternately aspirated from the cardiotomy reservoir and injected into the catheter.

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Table 1. Experimental Design

Dog No.	Left Kidney	Right Kidney	Day Killed
1	MCH-blood, filtered	Blood, filtered	5
2	MCH-blood, filtered	Blood, filtered	5
3	MCH-blood, filtered	Blood, filtered	3
4	MCH-blood, filtered	Blood, filtered	3
5	MCH-blood, filtered	Blood, filtered	2
6	Not accessible	Contrast medium	2
7	Not accessible	MCH-blood, no filter	2
8	Clots, no filter	Not accessible	2
9	MCH-blood, filtered	Blood, filtered	0[a]
10	MCH-blood, filtered	Not accessible	0[a]

[a] These animals were used in the pilot study.

MCH = microfibrillar collagen hemostat.

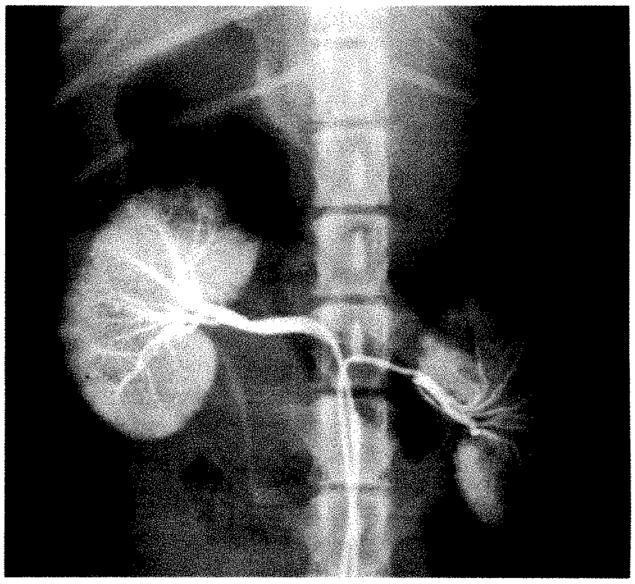

Fig 1. Angiographic study of dog 1 before autotransfusion. Ventrodorsal view of catheter placement in both renal arteries.

Before use, the autotransfusion system was primed with isotonic saline containing 10% acid citrate dextrose (vol/vol).

In Vivo Experiment

Transfusion of autologous blood was performed in 10 beagle dogs of either sex. They were 1 year old and weighed between 10 and 15 kg. Two of the animals were killed in a pilot study to evaluate the experimental design (Table 1). Before autotransfusion, each of the remaining 8 animals received 50 U/kg heparin intravenously. In the fully anesthetized dogs (anesthesia induced with oxymorphone, atropine, and thiopental and maintained with isoflurane) cutdown procedures were performed to expose the external carotid artery on one side and both femoral arteries. The carotid artery was catheterized and used to collect blood by controlled exsanguination into a sterile stainless steel vessel. Under fluoroscopy, a catheter ("Whithe" preformed catheter for pediatric angiography, 4.9F, renal curve; Beckton Dickinson, Rutherford, NJ) was advanced via the femoral arteries into the left and right renal artery, respectively. The positioning of the catheters and adequate perfusion of the renal vascular bed were evaluated angiographically (Hypaque-76; Winthrop-Breon Laboratories, New York, NY) (Fig 1).

Dogs 1 through 5 had transfused into one kidney blood that was collected from the carotid artery, mixed with MCHs (0.1 g MCH/100 mL shed blood), and passed through the filtered cardiotomy reservoir. The opposite kidney received blood collected and filtered in the same way but without MCHs. Fifty percent of the total blood volume of each dog (400 to 600 mL) was transfused into either renal vasculature over a period of approximately 30 minutes.

The dogs showed considerable anatomical variation of the renal arteries [18], preventing the simultaneous bilateral catheterization in 3 of the 8 surviving animals (Table 1). In these 3 dogs, two smaller arteries instead of one larger vessel branched off the aorta on one side. Thus the renal vasculature of only one kidney was accessible for perfusion. These 3 animals served as additional controls and were perfused in the following way into the accessible kidney. Dog 6 received contrast medium only, dog 7 was perfused with shed unfiltered blood containing MCHs, and dog 8 received unfiltered blood containing spontaneously formed blood clots.

Animals were allowed to recover after the procedure. Dogs 1 and 2 were killed on day 5 after operation, dogs 3 and 4 on day 3, and dogs 5 through 8 on day 2 (Table 1).

Blood samples were taken from each animal before the procedure and at regular intervals thereafter. The following variables were recorded (Fig 2): red blood cell count, hematocrit, platelet count, white blood cell count, total plasma protein, and plasma hemoglobin.

At necropsy each kidney was sectioned sagittally, and multiple sites of each kidney were taken for histological evaluation. The formalin-fixed sections of each kidney

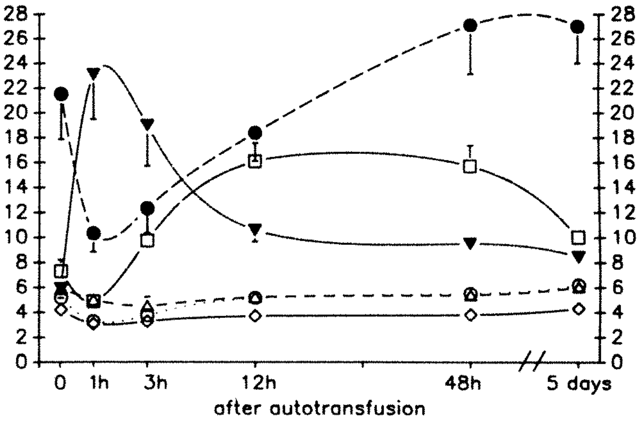

Fig 2. Hematologic variables, shown as mean ± standard error of the mean. (◇——◇ = hematocrit [×10%]; ○ · · · ○ = total plasma protein [g]; △---△ = red blood cell count [×10^6]; □——□ = white blood cell count [×10^3]; ●---● = platelet count [×10^4]; ▼–·–▼ = plasma hemoglobin [mg/dL].)

were processed in a routine manner and embedded in Tissue Prep 2 (Fisher Scientific, Fair Lawn, NJ). Sections 3-μm thick were cut on an LKB microtome (LKB 2218 Historange microtome; LKB, Stockholm, Sweden). All sections were stained with hematoxylin and eosin. Special stains (Mallory's phosphotungstic acid hematoxylin and immunohistochemistry using the avidin-biotin-peroxidase complex technique [19] for immunoglobulins G and C_3) were performed on selected specimens as required. Individual samples of raw MCH material were processed simultaneously to allow morphological comparison with MCH particles in renal tissues. Sections were also evaluated using polarized light microscopy.

In Vitro Experiment

In vitro tests were undertaken to assess the filter capacity of the cardiotomy reservoir. At a temperature of 37°C, 1,000 mL of Ringer's solution was mixed thoroughly with 1 g of MCH. The suspension was passed through the cardiotomy filter and the filtrate was evaluated for its MCH content. Aliquots were sampled before and after filtration. Batches were centrifuged for one hour at 10,000 *g* and pellets were examined histologically. Other batches were used to assess collagen quantitatively using a hydroxyproline assay [20]. Collagen types present in the used MCHs were analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis gel chromatography [21]. All in vitro experiments were triplicated and results given represent the statistical mean.

Results

All dogs survived the autotransfusion procedure without complication. Measurements of corpuscular blood variables, total plasma protein, and plasma hemoglobin of dogs 1 through 5 are shown in Figure 2. An insignificant decrease of red blood cell count, hematocrit, and total plasma protein was observed during the first two days after autotransfusion, with levels returning to normal between the third and fourth day. White blood cell count increased during the first two postsurgical days by 80%. Plasma hemoglobin level rose sharply to four times normal levels within the first two days and returned over the following two days to near normal. Platelet numbers decreased markedly within the first 24 hours and rebounded over the remaining time of observation to a level of 20% above their preoperative value.

At necropsy, none of the dogs perfused with filtered autologous blood (dogs 1 through 5) had notable macroscopic changes in either kidney. The kidneys of dog 6, perfused only with contrast medium, were normal. Dog 7, perfused with unfiltered blood containing MCHs, had multiple renal infarcts. Dog 8, transfused with autologous, partially clotted blood, showed only scattered areas of infarction.

Kidneys perfused with filtered blood alone showed uniform results. Occasional emboli up to 100 μm in diameter could be found in interlobular arteries, afferent arterioles, and glomerular capillaries. However, inflammatory perivascular infiltrates were sparse or absent, and there was no evidence of any degenerative changes in the tubular epithelial cells.

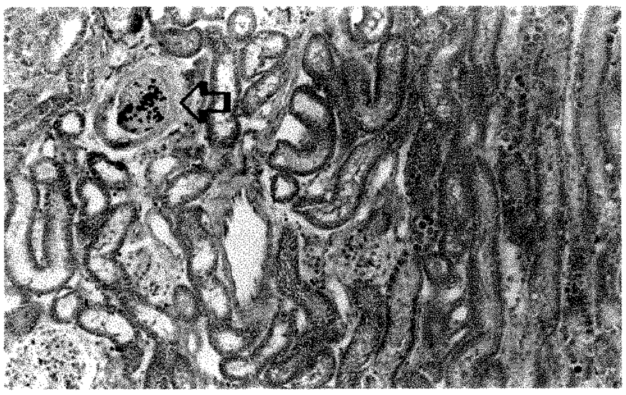

Fig 3. Intraarterial thrombus (arrow) *and selective acute tubular necrosis from dog 5, left kidney, obtained two days after transfusion with filtered, MCH-containing blood. Low-power photomicrograph, hematoxylin-eosin stain.*

Kidneys perfused with filtered blood mixed with MCHs had lesions that were related to the survival time after transfusion. Dogs killed on the second and third day after transfusion showed selective, multifocal acute tubular necrosis. In the affected tubules the nuclei were pyknotic and the cell cytoplasm was more brightly eosinophilic. Desquamation of epithelial cells into the tubule lumen was evident. Occasional intraarteriolar emboli containing an MCH foreign body were found (Fig 3). Polarized light microscopy confirmed these findings. Dogs killed on day 5 showed a severe multifocal tubule necrosis associated with severe arterial lesions. The vascular lesions ranged from a severe degenerative and necrotizing arteritis (Fig 4) to a fibrinoid necrotizing arteritis that morphologically resembled an immune-mediated arteritis (Fig 5). However, using immunohistochemical techniques, no distinct staining pattern was found.

Similar, yet far more intense histological changes were seen in the renal vasculature of the control animal transfused with unfiltered MCH particles. No evidence of microthrombus formation was found in the control dog who underwent renal angiography only.

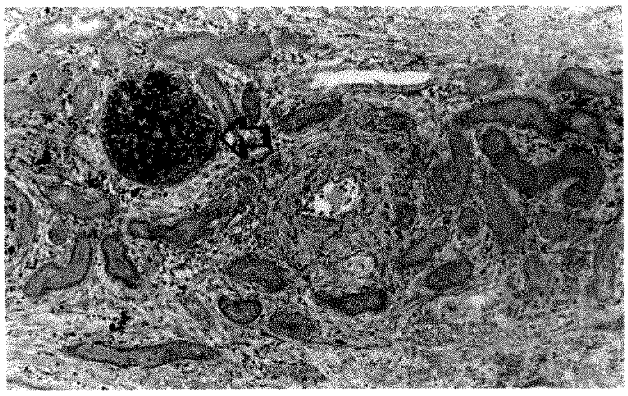

Fig 4. Degenerative and necrotizing arteritis (arrow), *tubule necrosis, and early interstitial fibroplasia from dog 1, left kidney, obtained five days after transfusion with filtered, MCH-containing blood. Low-power photomicrograph, hematoxylin-eosin stain.*

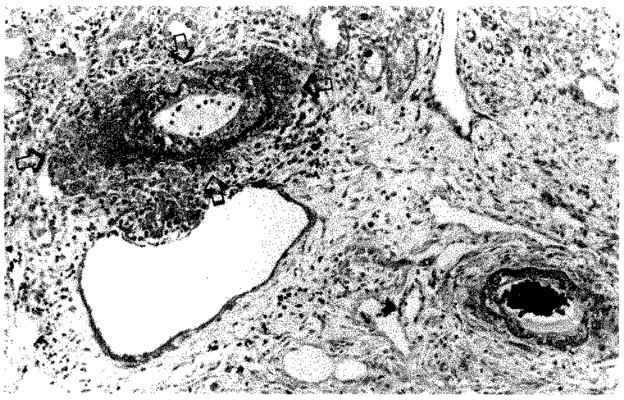

Fig 5. Fibrinoid necrotizing arteritis (arrows) *from dog 2, left kidney, obtained five days after transfusion with filtered, MCH-containing blood. Low-power photomicrograph, hematoxylin-eosin stain.*

By in vitro tests, the microfibrillar collagen hemostats consisted grossly of bovine collagen type I. No major protein contaminants could be detected. A moderate amount of collagen fibrils, some up to 200 μm in length, were found in the filtrates histologically. Hydroxyproline measurements of the MCH saline suspension before and after filtration showed that the filter removed approximately 98% of MCH particles. Mean collagen content in the unfiltered MCH saline suspension was 625 μg/mL as compared with 15 μg/mL collagen in the filtrate.

Comment

Our in vitro experiments showed that approximately 2% of MCH particles passed through the 20-μm cardiotomy reservoir filter (98% filter efficacy). An earlier study reported a 94% efficacy of filtration using the same MCH material in a 40-μm pore size filter system [13]. Though the filtering capability for MCH particles can be improved by reducing the pore size from 40 to 20 μm, the data suggest that fibrillar MCH particles still accumulate in noticeable amounts in filtered blood.

The in vivo studies show at the tissue level the effects of MCH particles passing in small numbers through the micropore filter. Microfibrillar collagen hemostat fibrils lodging in the renal vasculature were found in low numbers in sections of kidneys transfused with blood mixed with MCHs before filtration. In specimens of dogs killed two to three days after receiving renal perfusions, the degree of cellular reaction to the intravascular fibrillar debris and the surrounding microthrombi was moderate; on sections of animals killed five days after renal transfusion, strong inflammatory reactions involving the end-arterial vasculature, glomeruli, and renal tubules were seen. Although necrotizing endarteritis in combination with a lymphoplasma cellular infiltrate can be suggestive of an immune-mediated inflammatory reaction, the absence of a distinct staining pattern for immunoglobulins G and C_3 does not support such an assumption. Thus, most likely, the severe inflammatory responses present were elicited by the intraarterial MCH foreign bodies in combination with thrombotic vascular occlusion.

The high degree of multifocal inflammation in the blood-MCH–perfused kidneys is noteworthy and exceeded expectations because the number of filtered MCH particles trapped in the renal circulation seemed relatively low. However, it is possible that the number of MCH particles trapped in microthrombi was actually higher than estimated when the serial sections were evaluated.

A systemic mechanism eliciting strong vascular responses to relatively small amounts of foreign material was not likely to be involved. Hypertensive vascular necrosis based on systemic hypertension due to renal hypoperfusion is not likely to have developed in the previously normotensive dogs. Malignant hypertension is not elicited by unilaterally impaired renal blood flow, and the vascular lesions found in the affected kidneys were not characteristic for arteritis nodosa [22]. The disseminated nature of the inflammatory response might rather be explained by the formation of multiple emboli triggered by a few single foreign body emboli.

Although the pore size of the filter is only 20 μm, collagen fibrils with diameters of less than 20 μm but as long as a few hundred micrometers apparently can slide lengthwise through the filter pores as shown in our in vitro studies. These long filaments may also contribute to the more extensive vascular damage.

The hematologic variables measured before and after the experiment reflect reversible changes commonly seen in experimental autotransfusion in dogs [23, 24]. The drastic depletion of platelets seemed to be attributable to the length of the autotransfusion procedure, to the extracorporeal admixture of MCHs to the blood, and to platelet aggregation in the filtering device. Comparably, the number of platelets aggregated in renal microthrombi seems small.

Quantitatively, the amount of MCH particles passing through a filtered IAT device will generally be low. In most clinical settings, collected and filtered blood will be administered intravenously. Smaller amounts of MCH than used in this experimental study will reach the lungs in such transfused human patients and, most likely, the effect on pulmonary circulation will be substantially less than on the renal end-arterial vasculature of dogs. Intraarterial infusion is sometimes used in cardiac operations, however, while the arterial perfusion cannula is in place. Whatever return route of collected blood might be chosen, our findings emphasize the inherent danger of simultaneous use of MCH and IAT.

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Comparison of Ventricular Septal Surgery and Mitral Valve Replacement for Hypertrophic Obstructive Cardiomyopathy

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We have reviewed the results of two different forms of surgical management of hypertrophic obstructive cardiomyopathy refractory to medical therapy. Twenty-one patients were treated with 22 procedures between 1963 and 1987. Eleven underwent a ventricular septal procedure by myotomy with or without myectomy, and 11 underwent mitral valve replacement (MVR), 1 of whom had previously undergone a ventricular septal procedure. The groups were comparable with respect to severity and duration of symptoms, age range, electrocardiographic features, and hemodynamic changes. Mitral valve replacement produced a greater and more consistent reduction in the left ventricular outflow tract pressure gradient than a ventricular septal procedure (MVR, 68.3 mm Hg preoperatively and 2.5 mm Hg postoperatively; ventricular septal procedure, 60.1 mm Hg preoperatively and 13.4 mm Hg postoperatively). This was associated with better postoperative ventricular configuration in diastole and more apparent loss of midcavity narrowing in systole. Ejection fraction did not fall after a ventricular septal procedure but decreased significantly from a mean of 79% to a mean of 61% after MVR. Similarly, left ventricular end-diastolic pressure remained unchanged after a ventricular septal procedure but fell from a mean of 26.6 mm Hg to 17 mm Hg after MVR. Although both groups experienced a generally satisfactory symptomatic result, this appeared more reliable with MVR. We suggest that MVR offers a more predictable improvement than a ventricular septal procedure and may be the procedure of choice for units with limited experience with ventricular septal procedures.

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Most patients with hypertrophic obstructive cardiomyopathy (HOCM) can be satisfactorily managed by medical therapy with calcium-channel and β-adrenergic blocking drugs [1]. A minority, however, fail to respond adequately, and for these, a number of surgical approaches have been proposed, principally septal myotomy or myectomy, or both, and mitral valve replacement (MVR). Available information relates predominantly to septal procedures with relatively few published accounts of the results of MVR. There are no controlled trials that compare the results of these two forms of operative intervention and, given the problems inherent in attempting a trial with such an uncommon condition, it is unlikely that this information will ever become available.

Our experience with surgical management of HOCM includes equal use of ventricular septal procedures and MVR. We therefore reviewed the results obtained using these two techniques during the period 1963 to 1987 to provide comparative data regarding the outcome of these two entirely different surgical strategies.

Material and Methods

Surgical intervention was undertaken for HOCM in 21 patients during the period 1963 through 1986. All had primary HOCM with no other unrelated cardiac conditions. There were 10 male and 11 female patients with an age range of 12 to 68 years (mean age, 49 years). All had severe symptoms, ie, angina, palpitations, or syncope, that had failed to respond satisfactorily to medical treatment with β-adrenergic blocking drugs.

All patients were investigated by clinical examination, electrocardiography, and cardiac catheterization. Patients seen later in the series underwent echocardiographic assessment also. Repeat catheterization was undertaken in 15 of the 20 operative survivors at 2 to 5 years after operation. The 5 patients who were unwilling to undergo repeat catheterization were assessed by echocardiography. All patients were reviewed regularly at outpatient clinics. Follow-up ranged from 2 to 27 years (mean follow-up, 8.2 years) and was longer for patients who had a ventricular septal procedure (mean follow-up, 10.1 years) than for those who had MVR (mean follow-up, 5.4 years).

Cardiac catheterization was performed by the retrograde femoral route using transseptal venous catheterization for simultaneous readings in the inflow tract of the left ventricle, the ascending aorta, and the left ventricular outflow tract (LVOT). Mean pressure gradients were averaged over 30-second periods by analog computer.

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Cardiac output measurements were made in duplicate using indocyanine green dilution curves, and left ventricular regurgitant fraction was assessed by left atrial sampling [2]. Induced ventricular ectopic beats and isoproterenol provocation were employed to confirm LVOT obstruction when the resting gradient was small except for patients on β-blockade therapy (when these techniques were not used solely to enhance the gradient if the diagnosis was otherwise considered evident). Left ventricular angiography in the right anterior oblique projection was performed and simultaneous left and right ventricular angiography in the left anterior oblique projection was used to assess septal hypertrophy and systolic thickening and cavity encroachment. Except for 2 very young patients in whom coronary arteriography was considered unnecessary, this was performed in all patients, and normal vessels were demonstrated. Ejection fraction was calculated using the area–length formula for ventricular volume [3].

After 1970, M-mode echocardiography [4] was performed, and after 1980, this was supplemented by cross-sectional echocardiography using a 2.5- or 3.5-MHz transducer.

Results of treatment were assessed on the basis of relief of symptoms, absence of murmur, electrocardiographic (ECG) regression, residual LVOT pressure gradient, evidence of mitral reflux, and continued LVOT obstruction on angiography or echocardiography. Postoperative studies were limited in some cases to left heart catheterization studies if the patient was unwilling to undergo more extensive investigation. In those refusing cardiac catheterization, echocardiographic assessment was obtained.

All procedures were performed using cardiopulmonary bypass and moderate systemic hypothermia of 26° to 28°C. Ascending aortic and bicaval cannulation was employed. After 1976, myocardial preservation with cold crystalloid cardioplegia was used; before this, the heart was fibrillated and coronary perfusion maintained by direct ostial cannulation. Septal surgery, either a splayed myotomy (6 patients) or combined myotomy and myectomy (5 patients), was the only approach used before 1976. After the description of MVR by Cooley and colleagues [5] in that year, MVR was then also used.

Surgical treatment was divided equally between septal procedures (11 patients with a mean age of 49.4 years) and MVR (11 patients with a mean age of 52.1 years), although more septal procedures (7) than MVR procedures (4) were carried out in the precardioplegia era. One patient initially underwent a ventricular septal procedure and then MVR some years later because of recurrent LVOT obstruction. Prostheses implanted in the patients who survived MVR included six tilting-disc prostheses (29 and 31 mm) and four biological prostheses (27, 27, 31, and 31 mm).

Results

Early and Late Survival

There was one operative death in each group (9.1%). One patient undergoing septal myectomy before the use of cardioplegia died intraoperatively of ischemic cardiac contracture, which prevented the cessation of bypass. One patient undergoing MVR received a Björk-Shiley spherical-disc valve, which became jammed by a tertiary chordal fragment after bypass had been discontinued. Bypass was rapidly restarted and the valve exchanged for a porcine bioprosthesis. However, despite complete cardiac recovery, the period of reduced cardiac output had caused brain death. The patient died in the early postoperative period without regaining consciousness.

One late death (5%) occurred during follow-up. The patient had undergone MVR. Six years later, prosthetic valve endocarditis developed, which was inadequately treated with antibiotics.

Symptomatic Results

Review of the preoperative and postoperative New York Heart Association gradings indicated generally satisfactory improvement with both techniques yielding functional gains of between one class and three classes (mean gain with MVR, 1.9 classes; mean gain with septal procedure, 1.8 classes). Detailed patient review, however, indicated that there was a difference in the consistency with which good results were obtained.

Only 2 patients (18.2%) in the ventricular septal procedure group experienced complete and lasting relief of symptoms. Six (54.5%) experienced considerable symptomatic relief but required continuing β-blocker therapy. The remaining 2 survivors had poor results. One had recurrence of symptoms a year postoperatively, and his condition deteriorated over the subsequent 5-year period. Repeat catheterization demonstrated severe residual LVOT obstruction. Mitral valve replacement was then performed with complete relief of symptoms. Another patient underwent a repeat septal procedure within 12 months because of persisting symptoms; the symptoms were considerably improved but not eliminated.

All patients undergoing MVR had early and complete relief of symptoms, though 1 patient experienced recurrence of effort angina at 12 months. Continuing distal left ventricular systolic cavity obliteration was discovered at repeat cardiac catheterization and his symptoms were adequately controlled with β-blockade therapy. Another patient with severe supraventricular dysrhythmias preoperatively continued to sustain palpitations, though with decreasing frequency, postoperatively. Medication with amiodarone was therefore continued.

Clinical and Electrocardiographic Findings

Systolic ejection murmurs persisted after operation in 9 of the 10 surviving patients who underwent a septal procedure, whereas a murmur was present in only 1 patient after MVR. This patient had an apical systolic murmur and was known to have a mild mitral paraprosthetic leak. Regression of ECG evidence of left ventricular hypertrophy occurred in 4 patients, 1 who had a septal procedure and 3 who had MVR. Left bundle-branch block occurred in 2 patients in each group.

Catheterization and Echocardiographic Studies

Eight patients who had a septal procedure underwent both preoperative and postoperative catheterization. Their resting systolic LVOT pressure gradient fell from a

Table 1. Preoperative and Postoperative Hemodynamic Data for the Ventricular Septal Surgery Group

Patient No.	Operation	Status	Mitral Reflux	LVEDP (mm Hg)	CI (L/min/m^2)	EF (%)	LVOT Gradient (mm Hg)
1	Myotomy	Preop	No	8	2.5	. . .	88
		Postop	Yes	10	2.9	. . .	24 (46 provoked)
2	Myotomy	Preop	Yes	10	2.6	73	115
		Postop	Yes	29	1.9	86	27 (90 provoked)
3	Myotomy	Preop	Yes (moderate)	20	3.2	67	45
		Postop	No	28	1.9	59	0 (60 provoked)
4	Myotomy	Preop	Yes	30	1.3	75	36
		Postop	No	32	. . .	80	0 (0 provoked)
5	Myotomy	Preop	Yes	27	1.3	83	38
		Postop	No	20	1.4	90	3
6	Myotomy	Preop	Yes	20	2.2	79	24 (170 provoked)
		Postop	No	13	2.8	76	6
7	Myectomy	Preop	No	25	2.8	. . .	175
		Postop	Operative death	. . .	. . .	. . .	. . .
8	Myectomy	Preop	No	20	1.6	75	45
		Postop	Yes	15	1.9	89	35 (120 provoked)
9	Myectomy	Preop	Yes	24	2.6	73	90 (200 provoked)
		Postop	No	17	. . .	62	12
10	Myectomy	Preop	Yes	33	3.3	81	90
		Postop	Noninvasive review	. . .	. . .	. . .	. . .
11	Myectomy	Preop	Yes	10	. . .	. . .	160
		Postop	Noninvasive review	. . .	. . .	. . .	. . .

[a] Reflux was mild unless otherwise noted.

CI = cardiac index; EF = ejection fraction, LVEDP = left ventricular end-diastolic pressure; LVOT = left ventricular outflow tract.

mean preoperative value of 60.1 mm Hg (range, 24 to 115 mm Hg) to a mean of 13.4 mm Hg (range, 0 to 35 mm Hg). Similar data were available for 6 MVR patients. The mean preoperative LVOT gradient of 68.3 mm Hg (range, 20 to 110 mm Hg) fell postoperatively to a mean of 2.5 mm Hg. All but 1 of the MVR patients, however, had complete abolition of the gradient and the one remaining gradient measured only 15 mm Hg.

Preoperative and postoperative left ventricular end-diastolic pressure values were available for 8 patients who had a septal procedure and 7 patients who underwent MVR. The pressures were unchanged in the former group with a mean preoperative value of 19.9 mm Hg (range, 8 to 30 mm Hg) and a mean postoperative value of 20.5 mm Hg (range, 10 to 32 mm Hg). For the MVR patients, on the other hand, there was a reduction from 26.6 mm Hg (range, 14 to 38 mm Hg) to 17 mm Hg (range, 5 to 30 mm Hg). In general, therefore, MVR produced a more consistent result both in terms of predictability of gradient abolition and reduction of left ventricular end-diastolic pressure. Catheterization data are summarized in Tables 1 and 2.

Striking changes in the end-diastolic angiographic or echocardiographic size and shape of the left ventricular cavity were observed after both forms of intervention. However, they were more noticeable in the MVR patients, most of whom regained a more normal ellipsoid cavity shape and lost the systolic midcavity "waisting" present preoperatively (Figs 1, 2). After a ventricular septal procedure, the ejection fraction did not change. The mean preoperative value was 76% (range, 67% to 83%) and the mean postoperative value, 77% (range, 59% to 90%). There was, however, a significant reduction in the ejection fraction after MVR. The mean preoperative value of 79% (range, 64% to 89%) fell to a mean of 61% (range, 39% to 82%) ($p < 0.05$).

Comment

According to most series, at least two thirds of patients with HOCM are adequately managed by medical therapy [1, 6]. Refractory symptoms appear to be related to dynamic outflow tract obstruction and to diastolic dysfunction, but a proportion of patients exhibit substantial mitral reflux. Commonly, hypertrophy predominantly affects the interventricular septum, the left ventricular outflow tract, and the papillary muscles. Less frequently, there may be diffuse concentric hypertrophy of the entire

Table 2. Preoperative and Postoperative Hemodynamic Data for the Mitral Valve Replacement Group

Patient No.	Prosthesis	Status	Mitral Reflux[a]	LVEDP (mm Hg)	CI (L/min/m^2)	EF (%)	LVOT Gradient
1	Björk	Preop	Yes (moderate)	38	2.8	. . .	110
		Postop	No	15	1.9	. . .	0
2	Björk	Preop	Yes	13	1.9	. . .	50 (90 provoked)
		Postop	Noninvasive review	. . .	. . .	. . .	. . .
3	Björk	Preop	Yes	25	. . .	84	20
		Postop	Yes	9	2.3	56	0
4	Björk	Preop	Yes (moderate)	30	2.8	89	80 (>100 provoked)
		Postop	No	30	. . .	72	0
5	Ionescu	Preop	Yes	24	2.1	86	(130 provoked)
		Postop	No	5	. . .	71	0
6	Björk	Preop	No	27	1.8	. . .	140
		Postop	Operative death	. . .	. . .	. . .	. . .
7	Björk	Preop	Yes (moderate)	25	2.6	64	60 (140 provoked)
		Postop	No	25	. . .	46	0
8	Björk	Preop	Yes (moderate)	30	2.3	70	40
		Postop	No	25	. . .	62	0
9	Ionescu	Preop	Yes	22	2.4	73	30 (65 provoked)
		Postop	Noninvasive review	. . .	. . .	60[b]	. . .
10	Ionescu	Preop	No	14	4.4	82	100 (200 provoked)
		Postop	No	10	. . .	82	15
11	Wessex	Preop	Yes	25	1.9	87	100+
		Postop	Noninvasive review	. . .	. . .	39[b]	. . .

[a] Reflex was mild unless otherwise noted. [b] Value was not obtained by angiography.

CI = cardiac index; EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure; LVOT = left ventricular outflow tract.

left ventricle. Patients with either type have poor diastolic compliance. In the former, symptomatology is also related to LVOT obstruction due to anterior systolic motion of the anterior leaflet of the mitral valve and to bulging of the grossly thickened interventricular septum, which may partly obstruct right ventricular outflow also. Disorganization of the mitral subvalvular apparatus consequent to the hypertrophic process can lead to a greater or lesser degree of incompetence. Surgical management is concentrated on the LVOT obstruction with the goal of relieving either the septal hypertrophy or the abnormal mitral valve motion or both.

Surgical Techniques

A variety of techniques [7–16] have been employed to treat the LVOT obstruction in HOCM (Table 3). Other than the use of the valved apicoaortic conduit [16, 17] and aortoventriculoplasty [15], surgical management has concentrated on septal surgery by a variety of routes or MVR, or both. Taken overall, the transaortic route described and popularized by Morrow and colleagues [10, 18] is the most common of the surgical approaches currently used for HOCM. Operative mortality rates for septal procedures lie between 2% and 10% [6, 19–21] and are therefore similar to the 3.7% quoted by Cooley and associates [5] for MVR in this context and our own experience of 9% for both techniques.

VENTRICULAR SEPTAL SURGERY. The classic description of ventricular septal surgery is the myotomy-myectomy of Morrow and co-workers [10, 18], whose experience far exceeds that of any other group. There are many variants; some use differing routes to attack the muscle bulge, and others, like us, have considered that simply splaying a single myotomy to deepen and broaden the incision is adequate.

Although ventricular septal procedures have the obvious benefit of preservation of the native mitral valve (with authors claiming that mild mitral regurgitation will resolve after a satisfactory procedure), extensive sequelae have been described in association with this form of operation. Although it does not adversely affect patients who do not exhibit preoperative right bundle-branch block, it is noteworthy that left bundle-branch block

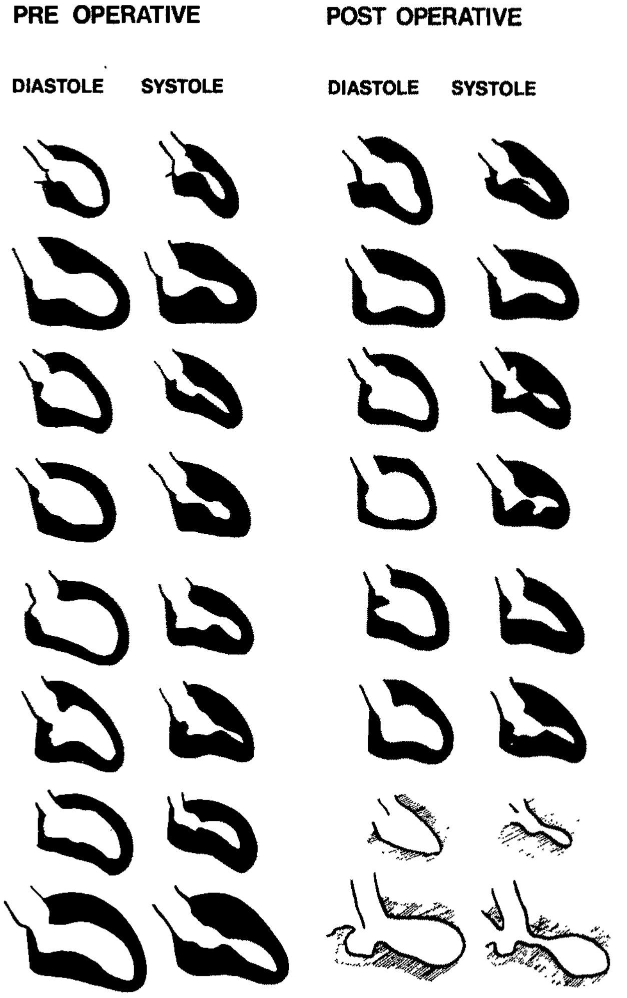

Fig 1. Preoperative and postoperative diastolic and systolic left ventricular cavity outlines (dense = *angiography;* hatched = *echocardiography) from 8 patients undergoing a ventricular septal procedure for hypertrophic obstructive cardiomyopathy.*

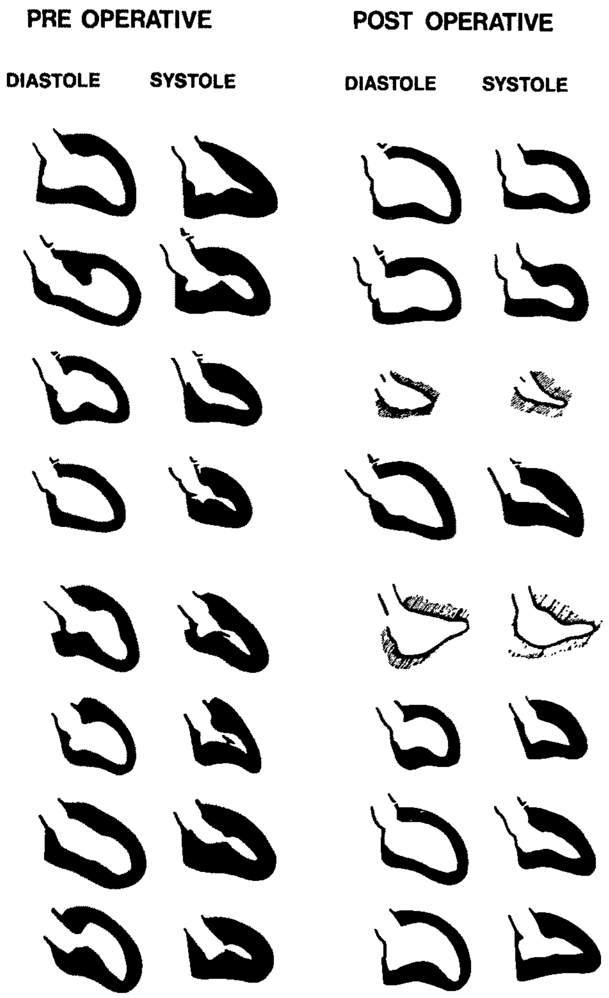

Fig 2. Preoperative and postoperative diastolic and systolic left ventricular cavity outlines (dense = *angiography;* hatched = *echocardiography) from 8 patients undergoing mitral valve replacement for hypertrophic obstructive cardiomyopathy. The ventricular cavity size is enhanced in diastole after mitral valve replacement, and there is reduced midcavity "waisting" in systole. These changes are less apparent after a septal procedure (see Fig 1).*

occurs in 30% to 70% of patients [18, 22], and in those in whom left bundle-branch block does not develop, left anterior hemiblock is usually present postoperatively [6, 22, 23]. Reported problems include aortic incompetence from damage to the retracted aortic leaflets at transaortic septectomy [21, 24], ventricular septal defect from perforation at the septectomy site, and mitral incompetence from inadvertent injury to the papillary apparatus when the myotomy cuts are performed [14, 24, 25]. Left ventricular aneurysm formation at the ventriculotomy site has been described with the transventricular approach [14, 22]. Apart from complications of the technique, which may themselves require operation, reoperation is occasionally needed for a recurrent gradient; Schulte and colleagues [25] quote a rate of 11%.

MITRAL VALVE REPLACEMENT. Although apparently technically straightforward, MVR, in fact, presents several problems in patients with HOCM. The implantation of a bioprosthesis in a young individual raises the prospect of a repeat procedure. Also, the small ventricular cavity can make implantation of a bioprosthesis difficult because of the relatively large strut apparatus, which may be more likely to become embedded in the ventricular muscle and thereby render subsequent repeat procedures more hazardous. The strut apparatus can itself give rise to LVOT obstruction in these patients, and, in our series, we have had to explant one bioprosthesis at the time of initial implantation and substitute a low-profile disc prosthesis for this problem. For these reasons, a mechanical valve is to be preferred, but high-profile mechanical prostheses are undesirable on the grounds that the tight fit within a small ventricular cavity could lead to valve dysfunction. It is therefore recommended [5] that a low-profile mechanical disc prosthesis be used. Disc impingement remains a concern in a small ventricular cavity, and as previously noted, 1 of our MVR patients died as a result of disc entrapment by a tertiary chordal fragment [26]. A mechanical prosthesis will, of course, necessitate permanent anticoagulation.

Table 3. Evolution of Surgical Techniques for Hypertrophic Obstructive Cardiomyopathy

Reference	Year	Technique
Brock [7]	1957	Closed transventricular myotomy
Cleland [8]	1958	Transaortic myectomy
Kirklin and Ellis [9]	1961	Combined transaortic and transventricular myectomy
Morrow and Brockenbrough [10]	1961	Transaortic myectomy
Lillehei and Levy [11]	1963	Trans left atrial myectomy
Harken [12]	1964	Trans right ventricular myectomy
Binet et al [13]	1968	Extended ventriculoaortic myotomy
Cooley et al [14]	1971	Mitral valve replacement
Rastan and Koncz [15]	1975	Aortoventriculoplasty
Dembitsky and Weldon [16]	1976	Valved apicoaortic conduit

Mitral valve replacement results in more than simply removal of the obstructing anterior mitral leaflet. It is likely that there is an element of ventricular stenting inherent in positioning a rigid prosthesis in the normally flexible mitral ring. This and division of the papillary muscles will cause opening of the ventricular cavity in diastole and reduce the contraction of the LVOT in systole. The effect of the papillary muscle division has been studied by intraoperative echocardiography during MVR and valvoplasty [27] with the findings of a 24% reduction in ejection fraction after valve replacement and papillary muscle section but not after valvoplasty.

Results of Operation for Hypertrophic Obstructive Cardiomyopathy

Those studies in which MVR has been compared with a ventricular septal procedure [28, 29] agree with our finding that gradient reduction is superior with MVR. Finghali and co-workers [29] demonstrated mean LVOT gradient reductions from 60 to 3 mm Hg after MVR, from 69 to 35 mm Hg after septal resection, and from 89 to 3.8 mm Hg with both together. Either technique, however, can produce excellent results. Extended follow-up of the patients of Morrow and associates [23] showed a reduction of the mean gradient postoperatively of approximately 90%, with 71% of postoperative catheterization studies revealing no residual basal gradient. In that large study of 240 patients, 88% showed improvement. Similar gradient reductions have been observed by others after septal resection [21, 22, 24]. All authors report good symptomatic improvement, with patients usually gaining one or two functional classes from a starting point in New York Heart Association class III or IV. Considerable improvement in exercise capability after operation has been reported, and this is superior to the benefit obtained using medical treatment [30].

One factor that may account in part for the considerably superior results we achieved with MVR compared with a ventricular septal procedure is that of familiarity. Apart from a few centers with a specialized interest in HOCM surgery, most general units see this condition infrequently. Certainly at our own rate of approximately one case per year, it would be difficult to accrue expertise in septal surgery. Mitral valve replacement, on the other hand, is a standard procedure in which all units are well versed. Therefore, some of the observed differences in outcome may be attributable to less well performed ventricular septal procedures. However, this does not detract from the excellent and predictable results of MVR, which may therefore be the procedure of choice for all nonspecialist units.

It can be argued that the more predictable surgical results of MVR will be obtained at the expense of the potential long-term complications of prosthetic valve replacement. With modern mechanical prostheses and good anticoagulation control, however, the risk associated with a prosthetic mitral valve may be a relatively minor adverse prognostic factor in the long-term outlook for patients having operation for HOCM.

Survival data following either form of surgery is not as reassuring as the symptomatic result. Finghali and colleagues [29], for example, reported an annual mortality of 3.8% for MVR and 2.0% for MVR and ventricular septal procedure combined. Although the annual mortality rates for ventricular septal procedures are usually 2% to 3% [21], Morrow and co-workers [23] quoted an annual mortality risk of 1.5% for their large series of patients having a septal procedure. Medical treatment alone, however, offers a worse outlook. Schulte and associates [25] described a 10% annual mortality with propranolol hydrochloride treatment alone on follow-up of 121 patients for a mean of 5.4 years compared with 2.5% for 80 patients having operation and followed for a mean of 4.2 years. Rothlin and colleagues [24] compared 63 patients who had operation with 49 who did not and found a 10-year survival of 80% and 71%, respectively, a difference that was not significant. However, the patients who underwent operation were symptomatically worse, had higher gradients, and had higher left atrial pressures. It is generally believed that the incidence of sudden death is probably not reduced [1], and this presumably is related to the persistence of a high rate of dysrhythmias [24].

Conclusion

Surgical intervention is indicated in those patients with demonstrable LVOT gradients and symptoms that do not respond adequately to β-blocker or calcium antagonist therapy. In these patients, operative intervention can be expected to produce a satisfactory reduction in gradient and increase in functional capacity. Both septal surgery and MVR can produce good results. Although the former has the advantage of avoiding a prosthesis, it has a range of serious complications of its own and probably requires a large accumulated experience for optimum results. We have noted superior cavity enlargement with MVR and therefore suggest that this form of therapy is certainly appropriate in patients with mitral dysfunction or very severe diastolic compliance problems. It is probably also indicated in patients with a thin basal anterior septum (<18 mm) or in whom the area of greatest septal thickening is difficult to access through a transaortic approach [31]. In that MVR is a routine surgical procedure and septal surgery is not, there may be an advantage in adopting MVR in nonspecialist units where the frequency of performing operations for HOCM would be low and the techniques of septal surgery would not be fully learned.

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INVITED COMMENTARY

Dr Walker and colleagues have made a valid observation that in centers that treat only an occasional patient with HOCM an MVR will produce a more predictable hemodynamic and clinical result than a left ventricular myotomy-myectomy (LVMM). It has also been our reported experience that MVR will more predictably relieve LVOT obstruction at rest and with provocation, decrease pulmonary arterial wedge pressure and left ventricular end-diastolic pressure, and relieve associated mitral regurgitation than will LVMM. The associated immediate complications of iatrogenic ventricular septal defects, complete heart block, and aortic or mitral insufficiency, which usually occur in 1% to 3% of patients undergoing LVMM, must be placed in perspective with the chronic complications occurring after MVR, including prosthetic endocarditis or thrombosis, thromboembolism, anticoagulation complications, and possible structural deterioration of a bioprosthesis. These potential complications are cumulative and ongoing after MVR, but once a successful LVMM has been performed, the iatrogenic risks cited are no longer risk factors. Patients treated by either operation are still at common risk for thromboemboli associated with atrial fibrillation and other dysrhythmias.

We continue to believe that patients having a septal dimension of 18 mm and the usual obstruction secondary to systolic anterior motion should undergo an LVMM, reserving MVR for those whose septum measures less than 18 mm or who have atypical septal morphology or a previous LVMM with persistent obstruction and symptoms. Mitral regurgitation, unless secondary to organic mitral valve disease and pulmonary arterial hypertension, is not an indication for MVR in our institution.

More recently, a combined LVMM and plication of the anterior mitral valve leaflet through the aortic annulus has markedly reduced MVR in our institution. The plication is limited to that area of contact of the anterior mitral valve leaflet that contacts the septum and is identified as a thickened area of scar. The extent of the mitral valve contact can also be appreciated by examination with intraoperative short-axis echocardiography, which aids in tailoring the plication to the individual patient. Postoperative studies in a limited number of patients have shown good relief of LVOT obstruction in most patients and no clinically significant mitral regurgitation or stenosis of the plicated mitral valve. We are encouraged by the early results of this adjunctive procedure to LVMM and hope that it will continue to be a possible way of avoiding MVR.

Dr Walker and colleagues also note that MVR is usually technically routine in patients with HOCM. This has been our experience as well in patients who do not have concentric left ventricular hypertrophy or massive hypertrophy of the interventricular septum. Technically, massive hypertrophy, a small left atrium, and a normal mitral valve annulus may add to the challenge of MVR in patients with HOCM. The use of pledget-reinforced sutures may help decrease the incidence of paravalvular leak. We feel that it is also important not to oversize the mitral prosthesis, as can be done in an arrested, flaccid heart, to avoid potential prosthetic malfunction. Finally, in view of the current trend to preserve the mitral valve apparatus when performing an MVR, this should not be done in patients with HOCM. The rigid ring of the mechanical prosthesis would fix the anterior mitral valve leaflet in the left ventricular outflow tract and obstruction from systolic anterior motion may still occur.

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High-Dose Aprotinin: Hemostatic Effects in Open Heart Operations

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Two groups of patients were prospectively studied to evaluate the hemostatic effects of high-dose aprotinin in open heart operations. Group A patients (n = 22) received aprotinin during the entire surgical procedure. Group B patients (n = 12) served as controls. The groups were homogeneous for base variables and for cardiopulmonary bypass duration. Postoperative bleeding was lower in group A (mean, 486 mL) than in group B (830 mL) ($p < 0.01$). The need for banked blood decreased by approximately half in the aprotinin patients (mean: group A, 213 mL; group B, 409 mL). Hemoglobin levels were similar in the two groups (postoperative day 7: group A, 11.29 g/100 mL; group B, 11.26 g/100 mL; NS). Platelet count decreased at the end of operation in both groups (99,000 and 95,000/mL, respectively; NS) and then increased beyond baseline levels before discharge. No complications were observed that could be attributed to aprotinin. In conclusion, we believe that the use of high-dose aprotinin is safe and effective. It decreases blood loss and reduces the need for banked blood in cardiac operations, particularly in select groups of patients (eg, those undergoing reoperation, Jehovah's Witnesses, those with renal failure).

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The need for blood transfusion in cardiac operations can be lessened with several techniques (eg, hemodilution, careful hemostasis, intraoperative and postoperative reinfusion of blood collected in the surgical field, autotransfusion) [1, 2]. Recently, a significant decrease in surgical bleeding was demonstrated with the clinical use of high-dose aprotinin [3–5]. In a prospective study we evaluated the effects of intraoperative infusion of high-dose aprotinin on bleeding and transfusion requirements.

Patients and Methods

Thirty-four consecutive patients (21 men, 13 women) aged 19 to 75 years (mean age, 59 years) underwent a cardiac operation. Written informed consent was obtained, and they were randomly divided into two groups according to birth date until an appropriate number of treated patients was reached. In group A patients (n = 22), at the start of anesthesia, 2,000,000 kallikrein inactivator units (KIU) of aprotinin (Trasylol, Bayer Leverkusen, FRG; 10,000 KIU/mL pure aprotinin with no additives) was infused over 20 to 30 minutes. Subsequently, 500,000 KIU/h aprotinin was given until the end of the operation. Additionally, 1,000,000 KIU of aprotinin was in the priming solution of the extracorporeal circuit. Group B patients (n = 12) did not receive aprotinin.

For all patients, autotransfusion was used if the basal hemoglobin level was greater than 12 g/100 mL. When appropriate, during the preoperative period as well as at the beginning of the surgical procedure, blood was collected for reinfusion after cardiopulmonary bypass (CPB).

The techniques of anesthesia and CPB were standardized. After premedication with diazepam (0.1 mg/kg), morphine (0.1 mg/kg), and scopolamine (0.3 mg), anesthesia was begun with fentanyl (25 to 35 μg/kg), diazepam (0.3 to 0.5 mg/kg), pancuronium bromide (0.1 to 0.2 mg/kg), and isoflurane 1%. Patients were ventilated with 50% oxygen and 50% nitrogen oxide. The extracorporeal circuit was made of a Stockert pump, a Shiley S100 bubble oxygenator, and Tygon tubes. Patients were heparinized before CPB with 300 U/kg, with additional doses to maintain the activated clotting time beyond 480 seconds. Heparin was neutralized after the end of CPB with protamine sulfate (1 mg/100 IU heparin). Operation was performed for heart valve disease in 20 patients, and for coronary artery disease in 14.

Plasma hemoglobin level, hematocrit, and platelet count were evaluated and other routine laboratory tests were performed before operation, after anesthetic induction, during and after CPB, at the end of operation, and on the first and seventh postoperative day.

The two groups were homogeneous with regard to base parameters (age, sex, weight, type of operation) and the duration of operation and CPB (Table 1). Preoperative defects of hemostasis and coagulation mechanisms were ruled out in all patients.

The amount of postoperative bleeding was evaluated, as well as the type of banked blood transfusion (eg,

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Table 1. Patient Data

Variable	Group A	Group B
Number of patients	22	12
Male	12	7
Female	8	5
Age		
Range	44–74	19–73
Mean	62 ± 1.4	57.8 ± 4.7
Weight (kg)	71.2 ± 2.5	69.8 ± 3.9
Coronary artery bypass grafting	9	5
Heart valve operation	13	7
Duration of operation (min)	311 ± 13	310.1 ± 27.5
Duration of cardiopulmonary bypass (min)	118.2 ± 7.3	118.7 ± 14.3
Autotransfusion (mL)	468 ± 62	576 ± 93
Hemoglobin (g/100 mL)	12.24 ± 0.39	12.20 ± 0.33
Platelet count (×10^8/L)	2.00 ± 0.13	2.10 ± 0.15

Where applicable, data are expressed as mean ± standard error of the mean. No statistically significant differences were seen between the two groups.

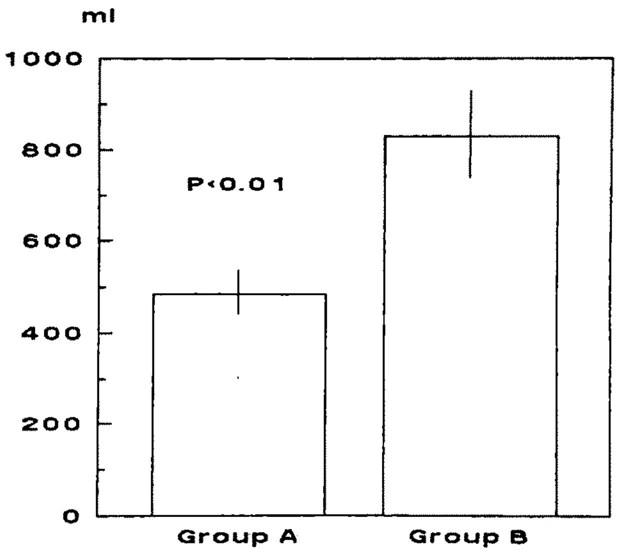

Fig 1. Postoperative blood loss (expressed in milliliters, ± standard error of the mean) was significantly (p < 0.01) less in patients treated with high-dose aprotinin.

packed red cells) needed during the hospitalization period. Banked blood transfusion was used only after reinfusion of autologous blood when patients needed volume expansion or had a hemoglobin concentration lower than 9 g/100 mL, or whenever hemoglobin fell to values below 8 g/100 mL.

One group B patient was excluded because of surgical bleeding that required reexploration for hemostasis.

For statistical analysis, Student's *t* test for independent samples was used to compare the means, and a significance level of 0.05 was assumed. When variances were not homogeneous, Welch approximation was used in calculating the appropriate degrees of freedom [6].

Results

Mean (± standard error of the mean) postoperative bleeding was 486 ± 47 mL in group A and 830 ± 117 mL in group B ($p = 0.00276$) (Fig 1). The average use of banked blood in group A was 213 ± 85 mL, approximately half as much as in the control group (409 ± 140 mL) (Fig 2). We did not compare transfusion figures statistically, as the use of blood did not depend exclusively on surgical bleeding, but also on hemodynamics and laboratory data. Thirteen patients in group A and 4 in group B did not receive homologous blood. The extent of bleeding and the need for transfusion, as well as all baseline variables and the length of cardiopulmonary bypass, were similar in the subgroups of patients who underwent coronary or valve operation.

The hematological perioperative data are reported in Table 2. The hemoglobin curve in the two groups is illustrated in Figure 3. The baseline mean values were 12.24 g/100 mL in group A and 12.20 g/100 mL in group B (not significant [NS]). During CPB, these figures decreased to 7.34 g/100 mL and 7.50 g/100 mL in groups A and B, respectively (NS). On postoperative day 7, the hemoglobin level was 11.29 g/100 mL in group A and 11.26 g/100 mL in group B (NS). The hematocrit curve paralleled the hemoglobin curve.

The platelet curve is shown in Figure 4. In the preoperative period the average number of circulating platelets was 2.00 × 10^8/L in group A and 2.10 × 10^8/L in group B. The platelet count dropped to 1.15 × 10^8/L in group A and 1.22 × 10^8/L in group B during CPB, and was as low as 0.99 × 10^8/L in group A and 0.95 × 10^8/L in group B at the

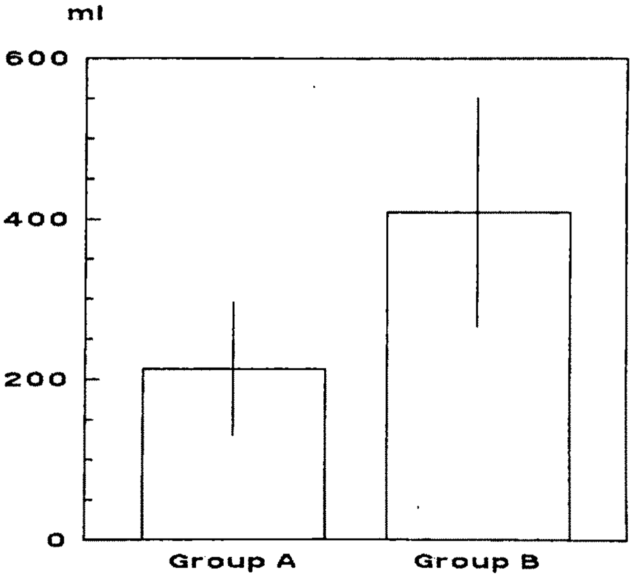

Fig 2. The average need for banked blood (expressed in milliliters ± standard error of the mean) was reduced in the aprotinin group.

Table 2. Hemoglobin and Platelet Curves

	Hemoglobin Level (g/100 mL)		Platelet Count (×10^8/L)	
Time	Group A	Group B	Group A	Group B
Admission (baseline)	12.24 ± 0.39	12.20 ± 0.33	2.00 ± 0.13	2.10 ± 0.15
Start of anesthesia	11.56 ± 0.32	11.07 ± 0.46	1.95 ± 0.13	1.89 ± 0.15
Cardiopulmonary bypass	7.34 ± 0.28	7.50 ± 0.33	1.15 ± 0.09	1.22 ± 0.21
After cardiopulmonary bypass	8.70 ± 0.34	9.12 ± 0.54	0.99 ± 0.07	0.95 ± 0.12
End of operation	11.09 ± 0.35	11.41 ± 0.50	1.22 ± 0.09	1.36 ± 0.15
First postoperative day	11.27 ± 0.35	10.38 ± 0.51	1.29 ± 0.08	1.43 ± 0.15
Seventh postoperative day	11.29 ± 0.30	11.26 ± 0.52	2.77 ± 0.19	2.70 ± 0.41

All values are given as mean ± standard error of the mean.

end of operation. In the postoperative period the platelet count increased again, reaching 2.77 × 10^8/L in group A and 2.70 × 10^8/L in group B (NS) on day 7.

We did not observe any complication due to the use of aprotinin. All patients were discharged between postoperative days 8 and 20.

Comment

A reduction in the use of homologous blood in cardiac operations is becoming increasingly important, due to the serious risks connected with transfusions (eg, hepatitis, acquired immunodeficiency syndrome) [4, 7, 8]. A decrease in bleeding and thus in the need for banked blood using high-dose aprotinin has been reported recently [3–5].

The exact mechanism by which aprotinin modulates the hemostatic system during and after CPB is not completely known [4, 8, 9]. During CPB, activation of a humoral system is observed, which determines protease release in the context of the so-called whole-body inflammatory reaction; these proteases may act on platelets and granulocytes [10]. Using high doses of aprotinin, kallikrein and plasmin are effectively inhibited and platelet function is thus preserved during CPB [5].

The data from this study show that the number of circulating platelets decreases during and immediately after CPB; it then increases to reach a value greater than the baseline on postoperative day 7. Though Van Oeveren and colleagues [5] demonstrated that in patients treated with aprotinin there was no platelet decrease in the early phases of CPB, we did not observe this behavior. Quantitatively we measured no statistically significant differences between the treated patients and the control group. We believe that only further studies on platelet function will clarify the real physiopathological mechanisms.

Some of the clinical studies concerning the use of high-dose aprotinin relate to reoperations [3, 4]. Only 4 of our patients had undergone a previous heart operation, and they showed a bleeding tendency not different from the group average; there was also no difference in mean extent of blood loss demonstrated between coronary and valve operation subgroups. The decrease in bleeding rates obtained in the group of patients treated with aprotinin was statistically significant ($p < 0.01$). We measured the total amount of postoperative bleeding because that is the

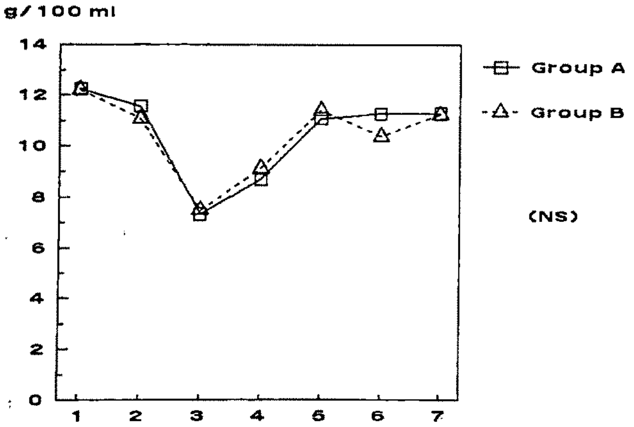

Fig 3. The hemoglobin curve through the perioperative period shows no statistically significant differences between groups A and B. (1 = hospital admission [baseline]; 2 = start of anesthesia; 3 = cardiopulmonary bypass; 4 = after cardiopulmonary bypass; 5 = end of operation; 6 = first postoperative day; 7 = seventh postoperative day.)

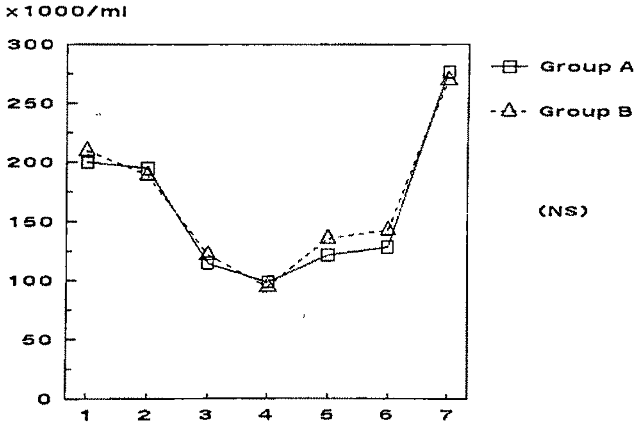

Fig 4. Platelet count curve was parallel in the two groups (no significant difference). (1 = hospital admission [baseline]; 2 = start of anesthesia; 3 = cardiopulmonary bypass; 4 = after cardiopulmonary bypass; 5 = end of operation; 6 = first postoperative day; 7 = seventh postoperative day.)

type of bleeding we could reliably measure; intraoperative bleeding is related to many different factors, such as heparinization and surgical hemostasis, which is obviously not complete until the chest is closed. In many patients treated with aprotinin, serum instead of blood was seen in the operative field before closure of the chest wall. In both groups the drainage catheters were routinely removed at the 36th postoperative hour. At this time, however, blood had stopped draining from the chest for at least 24 hours. We did not test the waste blood for hemoglobin concentration; however, the color of the drain collection was uniformly pale. Therefore, the total loss figures must be considered overestimated, particularly in the aprotinin group.

The need for banked blood was reduced by approximately half in the treated patients, but we would like to emphasize that the extent of transfusion was rather low in all our patients (mean, 213 mL in group A and 409 mL in group B). The beneficial effect of high-dose aprotinin in reducing the quantity of transfused blood with no change in plasma hemoglobin levels in the postoperative period is, however, of clear clinical value. Moreover, in 13 of 22 treated patients, we were able to use no homologous blood at all. From the statistical analysis of the data we observed that the use of autotransfusion did not bias any behavior within the groups.

We could not detect any contraindication to the use of aprotinin even at high doses—anaphylactic shock, allergic manifestations, or drug-related morbidity were never observed. On the other hand, aprotinin may be indicated not only for its hemostatic effects, but also for its preventive action on some of the aspects of the whole-body inflammatory reaction, such as pulmonary dysfunction, mediated by humoral factors that may be inhibited by aprotinin [5, 11].

In conclusion, we demonstrated that with the use of high-dose aprotinin during open heart operations, there is a significant reduction in postoperative bleeding and therefore a decreased need for banked blood. The hematologic parameters we studied were not negatively influenced and no complications related to this technique were observed. We believe that the clinical use of aprotinin can be particularly effective, and that it decreases the need for blood transfusion in select patients, such as those undergoing reoperation, those with renal failure, Jehovah's Witnesses, or those in whom extended CPB is expected. In these groups of patients, we decided to adopt high-dose aprotinin as a routine in our present clinical practice. Further studies are needed to extend the application of this technique to all patients in the future.

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How Soon Should Drainage Tubes Be Removed After Cardiac Operations?

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Pericardial effusion frequently occurs after cardiac operation. Despite its high incidence, the etiological process of postoperative pericardial effusion remains unclear. Residual blood or thrombus has often been suggested as a possible cause, implying that the occurrence of pericardial effusion could be related to the effectiveness of postoperative thoracic drainage. This possible relationship, however, has never been studied. We found that prolonging the duration of thoracic drainage by 24 hours often increases total chest tube output considerably but does not affect the incidence of postoperative pericardial effusion: approximately 55% of 100 patients in this study were shown by two-dimensional echocardiography to have pericardial effusion on the sixth postoperative day, regardless of the duration of postoperative drainage. Because of this, and because a long period of drainage causes discomfort for the patient, mechanical irritation to the heart and the pericardium, and an increased risk of infection, we recommend removing drains as soon as their efficacy has peaked, preferably on the first postoperative day.

(Ann Thorac Surg 1989;48:540–3)

Pericardial effusion (PE) frequently occurs after cardiac operations [1–4] and may cause hemodynamic embarrassment and, if chronic, prolong hospitalization. Progression of PE to tamponade, although rare [5–7], is a much feared complication after cardiac operations.

Although no consensus exists regarding the etiological process of PE, several groups of investigators have suggested that residual blood or thrombus in the pericardial space may be important factors [3, 7, 8]. Therefore, adequate removal of blood from the pericardial sac and the mediastinum after operation by drainage may be essential in attempting to restrain the process of PE. To accomplish adequate drainage, chest tubes must be properly placed and their patency must be ensured, and the tubes must remain in place for a sufficient amount of time. Thus, removing chest tubes too early may result in avoidable postoperative complications. However, a long period of drainage is known to cause discomfort for the patient, mechanical irritation to the heart and pericardium, and an increased risk of infection [9, 10].

A review of the literature showed that standard criteria to determine the moment when drains should be removed have not been established. In our clinic, chest tubes are preferably removed on the first postoperative day if drainage has been 50 mL or less during the previous four hours. If this condition is not fulfilled on the first postoperative day, drains are removed on the second postoperative day or even later. The present study compared patients in two groups to determine whether duration of drainage is related to incidence of postoperative pericardial effusion and thereby to contribute to establishment of clear criteria for drain removal.

Material and Methods

One hundred adult patients who underwent a cardiac operation at the Amsterdam Academic Medical Center between May and November 1988 were studied. This group consisted of 65 men and 35 women, ranging in age from 29 to 82 years (mean age, 62 ± 10 years). Potential subjects were excluded if (1) they had undergone a previous cardiac operation, (2) they did not undergo routine anticoagulation therapy, (3) they had serious postoperative complications such as severe myocardial failure, or (4) their chest tubes could not be removed within 24 hours after the operation because of persisting drainage. Subjects were randomly assigned to one of the following treatment groups: group 1 (n = 50), in which chest tubes were removed according to our usual conditions within 24 hours after the operation, and group 2 (n = 50), in which all patients routinely would have had their chest tubes removed within 24 hours after operation, but an additional period of drainage was effected in each of them by removing the drains on the second postoperative day, about 24 hours after this normally would have been done. The operations performed in each group are shown in Table 1.

Before cardiopulmonary bypass, heparin (2.5 mg/kg body weight) was administered to achieve an activated clotting time of at least 500 seconds. The effect of heparin was reversed by protamine sulfate. The cardiopulmonary bypass units were equipped with a Cobe CML-2 membrane oxygenator. The pump priming consisted of

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Table 1. Surgical Procedures Performed in Both Treatment Groups

Procedure	Group 1	Group 2
CABG	41	42
AVR	6	4
CABG + AVR	2	2
Mitral valve replacement	1	2

AVR = aortic valve replacement; CABG = coronary artery bypass grafting.

Table 2. Incidence of Pericardial Effusion Compared With Duration of Postoperative Drainage

Pericardial Effusion	Group 1	Group 2
None	24	21
Slight	17	17
Moderate	9	12
Heavy	0	0

Significance: $\chi^2 = 0.63$, $p > 0.3$.

Haemacel (1,500 mL), Ringer's lactate (500 mL), mannitol 20% (100 mL), and sodium bicarbonate 8.4% (50 mL). Each patient had a 32F tube placed retrosternally and a second 28F tube placed posteriorly in the pericardial space. If one or both pleural spaces were opened, additional tubes were inserted. In most patients, the upper two thirds of the pericardium was closed at 2-cm intervals. Each tube was connected to a disposable water seal drainage system with 10 cm H_2O suction. Chest tubes were frequently milked and stripped in the early postoperative period to ensure tube patency.

Anticoagulation with Coumadin (crystalline warfarin sodium) derivates was started on the morning of the first postoperative day in all subjects. Dosages were such that the patients' thrombotest attained a level between 5% and 15%. Subjects had not received anticoagulants before operation.

To assess the presence and size of PE, two-dimensional echocardiography was performed on the sixth postoperative day. Pericardial effusion was classified as follows: (1) slight, fluid was localized only at and below the atrioventricular groove, or thin traces of fluid were seen anywhere between epicardium and pericardium; (2) moderate, a fairly uniform distribution of more fluid was seen posteriorly and apically (Fig 1); and (3) heavy, fluid was present posteriorly, apically, and anteriorly.

Data were analyzed statistically by Student's t test and the χ^2 test. A Pearson product-moment correlation coefficient (r) was calculated where appropriate. Data are represented as the mean ± standard deviation. A p value of less than 0.05 was considered statistically significant.

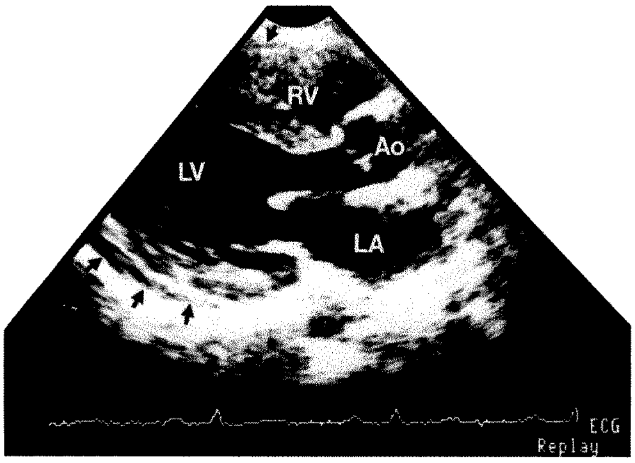

Fig 1. Parasternal long-axis view at the level of the base of the heart showing an example of a moderate pericardial effusion (arrows). Effusions markedly heavier than the one shown were not observed in any of the patients. (Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.)

Results

Basic Drainage Period

All 100 patients included in this study reached the point at which less than 50 mL was drained in a four-hour period within 24 hours after operation. (Thirty-one percent of all patients operated on during the study period who did not meet any of the three other exclusion criteria described had drainage persisting longer than this "basic" period and were excluded for this reason.) Mean chest tube output during the basic period was 661 ± 355 mL in patients without pleural chest tubes and 863 ± 445 mL in patients with pleural chest tubes ($p < 0.02$). Patients with aortic valve replacement alone or in combination with coronary bypass grafting demonstrated a significantly larger blood loss (860 ± 470 mL) during this period as compared with patients without aortic valve replacement (660 ± 357 mL) ($p < 0.05$).

Additional Drainage

Fifty patients had an additional 24-hour drainage period, which resulted in an increase in mean chest tube output. Output was also significantly greater in patients with pleural tubes (range, 0 to 355 mL; mean, 153 ± 100) than in patients without pleural tubes (range, 0 to 225 mL; mean, 97 ± 60) ($p < 0.02$). The large standard deviations reflect the striking variability associated with these values. The aspect of fluid drained in the additional period gradually became more serous in most patients. Statistical analysis showed that the amount of chest tube production in the additional period was not significantly correlated with blood loss in the basic period ($r = 0.12$, $p > 0.3$). Furthermore, additional chest drainage was not related to the patient's age, sex, weight, and type of operation performed. The duration of cardiopulmonary bypass was compared with chest tube output and showed a trend toward a positive correlation with drainage in both the basic ($r = 0.11$) and the additional ($r = 0.22$) drainage period. Neither trend, however, was statistically significant ($p > 0.15$).

Pericardial Effusion

Table 2 shows the incidences of postoperative PE. Clearly, incidence of PE was generally not affected by differences

Table 3. Relation of Pericardial Effusion to Amount of Chest Tube Drainage in the Additional 24-Hour Period

Additional Drainage	No PE	PE
≤60 mL	10	3
>60 mL	11	26

Significance: $\chi^2 = 6.96$, $p < 0.01$.
PE = pericardial effusion.

in duration of postoperative drainage. The effect of postoperative anticoagulation therapy, as measured with the thrombotest, had no correlation with the presence of PE. Furthermore, the incidence of postoperative PE appeared to be unrelated to the patient's age, sex, weight, or type of operation or to the presence of tubes in the pleural space.

The possible influence of the amount of chest drainage on PE was also evaluated. Chest tube output in the basic drainage period showed no correlation with PE on the sixth postoperative day. Table 3 compares the incidence of PE with the amount of chest drainage in the additional period. The less frequent occurrence of PE in patients who had less than 60 mL of additional drain production proved statistically significant. Delayed cardiac tamponade was not observed in any of the subjects.

Comment

Several echocardiographic studies have been undertaken to investigate the incidence of PE after cardiac operations. Reifart and colleagues [4] reported an incidence of 26%, whereas other investigators found PE in over 50% of operated patients [1–3]. Weitzman and colleagues [2] had the highest reported incidence: 85% of all patients in their study were found to have postoperative PEs. The variability between these estimates is probably partly caused by certain pitfalls in the ultrasound diagnosis of PE. Isolated anterior echo-free spaces can be especially difficult to interpret, as often not PE but adipose tissue or other material causes such spaces.

Despite the high incidence of effusion, associated delayed cardiac tamponade is a rare complication, occurring in 0.8% to 1.3% of all operated patients [5–7]. However, hemodynamic effects may also be present when clinical signs of tamponade are not. Intermediate effects of PE include an exaggerated inspiratory decrease in left ventricular filling and ejection time and an abnormal inspiratory increase in preejection period [11, 12]. The extent to which PE influences cardiovascular physiology mainly depends on the amount of fluid, the elasticity of the parietal pericardium, and the rate of fluid accumulation. Postoperative development of pericardial adhesions may also be a determinant, as it may cause loculation of pericardial fluid with subsequent tamponade [13]. Thus, complications of postoperative PE are very difficult to anticipate.

The cause of postoperative PE is most likely multifactorial. Residual blood or lysis of residual pericardial thrombus has been suggested to cause effusion by irritating the pericardium with hydrophilic action [7, 8], which could result in postoperative drainage. The possible correlation of drainage with PE is twofold. First, when chest tubes are removed too early, persistent slow bleeding may cause gradual accumulation of blood in the pericardial space. Second, more efficient removal of blood and clots by prolonging the duration of chest drainage may prevent irritation and hydrophilic action.

The best time to remove drains is difficult to determine; serosanguineous drainage seldom ceases completely. Information regarding drainage policy in postoperative care was obtained from all 12 Dutch cardiac surgery clinics. Six of them had no specific criteria for production before drain removal. The criteria of those that did varied substantially, ranging from 50 mL/h or less for three hours to 40 mL of total output in an eight-hour period. In one clinic, chest tubes were never removed earlier than the second postoperative day.

The possible relationship between the duration of postoperative drainage and the development of PE has never been studied. One study reveals that prolonging the duration of chest drainage by 24 hours can increase drainage of mostly serosanguineous fluid substantially, especially in patients in whom pleural tubes are inserted. The latter is a strong indication that the additional output is at least partly caused by the presence of the chest tubes themselves, as the pleura is very susceptible to mechanical irritation. The great variability in additional chest tube output, however, suggests that more factors are involved. Our efforts to explain why some patients have more than 200 mL of additional chest tube output whereas others produce practically nothing were unsuccessful. No variables significantly influenced additional chest tube output. In particular, the amount of "early" postoperative drainage had no predictive value.

In both treatment groups, the incidence of PE on the sixth postoperative day was about 55%. This finding supports the results of previous studies, in which similar incidences of postoperative PE were reported [1, 3]. None of the observed effusions was large, and no subject had severe hemodynamic consequences of PE.

Postoperative effusion was previously reported to occur more frequently in patients with large amounts of postoperative drainage [1]. In this study, PE correlated with drainage in another way. The occurrence of PE was unrelated to blood loss in the basic drainage period. However, patients who produced less than 60 mL in the extra 24-hour period had PE significantly less frequently than did patients with more than 60 mL of additional drainage. This is an important finding, indicating that in some patients the process leading to PE on the sixth postoperative day probably is already active on the first and second postoperative day.

There are several possible reasons why prolonged drainage does not cause a decrease in frequency of postoperative PE. Postoperative mediastinal drainage, regardless of its duration, never results in a complete evacuation of fluid from the pericardial space [14]. The undrained fluids may cause irritation or exert hydrophilic action or may themselves still be visible on echocardiography on the sixth postoperative day. In addition, postoperative PE and cardiac tamponade have been suggested to correlate with use of anticoagulants [15, 16]. That we routinely administer anticoagulants in patients who have undergone coronary artery bypass grafting, which is not common in the United States, may have had an effect on the overall incidence of PE in both groups. However, because Coumadin therapy started on the first postoperative day takes considerable time to reach its full effect, we believe the results of our study regarding the effects of prolonging drainage are unrelated to use of anticoagulants. Finally, traumatic and infectious pericarditis have also been suggested as possible causes of PE unrelated to postoperative drainage.

In summary, this study proves that prolonging postoperative thoracic drainage has no advantageous effect on the incidence of PE after a cardiac operation. We conclude that drains generally can be removed safely on the first postoperative day, as soon as production is less than 50 mL for four hours or perhaps even sooner. Removing chest tubes later unnecessarily increases the risk of infection [9, 10], mechanical irritation by the tubes, and discomfort to the patient.

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Intraoperative Evaluation of Atrioventricular Septal Defect Repair by Color Flow Mapping Echocardiography

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Despite improving survival rates after repair of atrioventricular septal defect, many patients require reoperation because of postoperative heart failure. We used intraoperative color flow mapping echocardiography to assess the results of surgical repair of atrioventricular septal defect in 19 consecutive patients and compared those findings with results three to five days and 3 to 11 months after repair. There was close correlation between intraoperative and postoperative color flow mapping echocardiography in estimating the presence and severity of left atrioventricular valve regurgitation. All patients survived surgical repair, but in 4 (21%), postoperative congestive heart failure due to left atrioventricular valve regurgitation developed. The need for reoperation was significantly correlated with the severity of left atrioventricular valve regurgitation ($r = 0.68$) as estimated by intraoperative echocardiography and preoperative aberrancies in the atrioventricular valve ($r = 0.68$). Age, weight, additional congenital heart disease, preoperative pulmonary vascular resistance, preoperative atrioventricular valve regurgitation, and postoperative mean pulmonary arterial or left atrial pressure were not significantly correlated with the need for reoperation. Intraoperative color flow mapping echocardiography can accurately predict the development of early postoperative heart failure and subsequent reoperation after surgical repair of atrioventricular septal defect.

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In the past 15 years, the surgical repair of atrioventricular (AV) septal defect has improved, with operative mortality rates decreasing from more than 50% [1, 2] to less than 10% [2–5]. After hospital discharge, a substantial number of patients with repaired AV septal defect will develop congestive heart failure that may be fatal. This late mortality and morbidity have been related to persistent pulmonary vascular obstructive disease and residual cardiac defects. Failure of left AV valve repair has been the usual cause of late symptoms. In one series [6], 53% of the late deaths were attributable to failure of the left AV valve repair. Reoperative rates for left AV valve failure have been as high as 24% [2].

Preliminary studies [7, 8] have demonstrated the feasibility of intraoperative color flow mapping (CFM) echocardiography as a means to determine the adequacy of surgical repair in congenital heart disease. Our aims in this study were (1) to compare the results of intraoperative CFM echocardiography after surgical repair of AV septal defects with studies performed early (three to five days) and late (3 to 11 months) after operation; and (2) to assess the relationships of CFM echocardiographic findings, surgical mortality and morbidity, and preoperative and postoperative anatomy and hemodynamics.

Material and Methods

Patient Population

Between December 1986 and October 1988, 19 consecutive patients with AV septal defect underwent intracardiac repair at St. Louis Children's Hospital. Four of these 19 patients had a restrictive ventricular septal defect and were operated on between 17 and 24 months of age. One of the remaining 15 patients was aged 11 years; the others underwent surgical repair between 2 and 13 months of age (mean, 5.9 months).

Seven of the 19 patients had other congenital heart disease (patent ductus arteriosus, 2; small additional muscular ventricular septal defects, 2; coarctation of the aorta, 1; infundibular and valvular pulmonary stenosis, 1; and double-outlet right ventricle, 1). Seventeen of the 19 patients had trisomy 21.

Preoperative Evaluation and Operation

All patients had CFM echocardiography, cardiac catheterization, and angiography before operation to assess pulmonary vascular resistance, anatomy, and AV valve regurgitation. Parasternal, apical, and subxiphoid echocardiographic views were used as previously described [9, 10] to evaluate the AV valve, its chordal attachments, and its papillary muscle distribution. The morphology and comparative size of the bridging and mural leaflets of the AV valve were determined at the time of operation by direct inspection. All patients had AV repair with the

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two-patch technique described by Carpentier [11], making the left AV valve a tricommissural valve.

The AV valve leaflets were attached to the crest of the ventricular septal patch with a nonpledgetted running suture technique, which was also supported by the running suture of attachment of the atrial patch to the ventricular patch and common AV valve. Ventricular distention was used during operative repair to assess the location of the valve incision and the level of attachment of the valve to the patch, and postoperatively to assess the competency of the left AV valve repair.

The commissure between the left superior and inferior leaflets was not closed if left AV valve competence appeared acceptable. When evidence of leakage on testing of the AV valve occurred at the commissure between the left superior and inferior leaflets, partial or near complete closure of this "cleft" was performed with interrupted fine monofilament sutures. When testing of the repaired left-sided AV valve was associated with continued central insufficiency or insufficiency at the remaining commissures between the left inferior and posterior leaflets or the left superior and posterior leaflets, localized annuloplasties were performed with interrupted pledgetted monofilament suture.

Color Flow Mapping Echocardiography

Between December 1986 and June 1987, studies of 3 of the 19 patients were performed on an Aloka SSD-880 system using a 3.5-MHz transducer. Thereafter, a Corometrics (Aloka) SSD-860 system was used with a 5.0-MHz or 3.5-MHz transducer depending on the patient's size. Studies were performed in the velocity variance mode with the standard color assignments for direction of flow, flow velocity, and flow variance. Pulse repetition frequencies varied between 6 and 8 MHz. B-mode scanning angle was kept at 90 degrees with a color flow angle at 45 degrees. The focal distance varied between 40 and 75 mm depending on the patient's size.

Color flow gain was adjusted so that white background noise was faintly present, similar to previous intraoperative studies [12, 13]. Contour edge enhancement, color enhancement, color edging, and color compressions were not used. Color flow filtration was kept in the "high" position. With these settings (Fig 1), lower velocity intracardiac flows are not visualized; thus, abnormal flows from septal defect or valvular regurgitation were highlighted.

Intraoperative echocardiograms were done from an epicardial position using a transducer covered with a sterile plastic sleeve. A cushion of ultrasound gel was placed between the transducer and the sleeve. All patients were in sinus rhythm and were receiving intravenous inotropic or vasodilating drugs to maintain blood pressures within the normal range for age during the studies.

Two transducer positions were used. In the first position (Fig 1A), the transducer was placed over the right ventricular outflow tract. In the second position (Fig 1B), the transducer was placed as near to the cardiac apex as possible. In each view, the transducer was rotated and angled in an attempt to visualize all the cardiac chambers and valves and their maximal dimensions. Total study time was approximately ten minutes. One study was eliminated from analysis due to transducer malfunction. Postoperative CFM echocardiograms were performed three to five days after repair. One of the patients was on a ventilator at the time of study. Color flow mapping echocardiograms were then repeated 3 to 11 months after operation. Conventional pulsed and continuous-wave Doppler echocardiography was used to confirm an abnormal flow disturbance in the region of an abnormal color flow pattern. No patient was sedated for these studies. The same physician interpreted each echocardiogram.

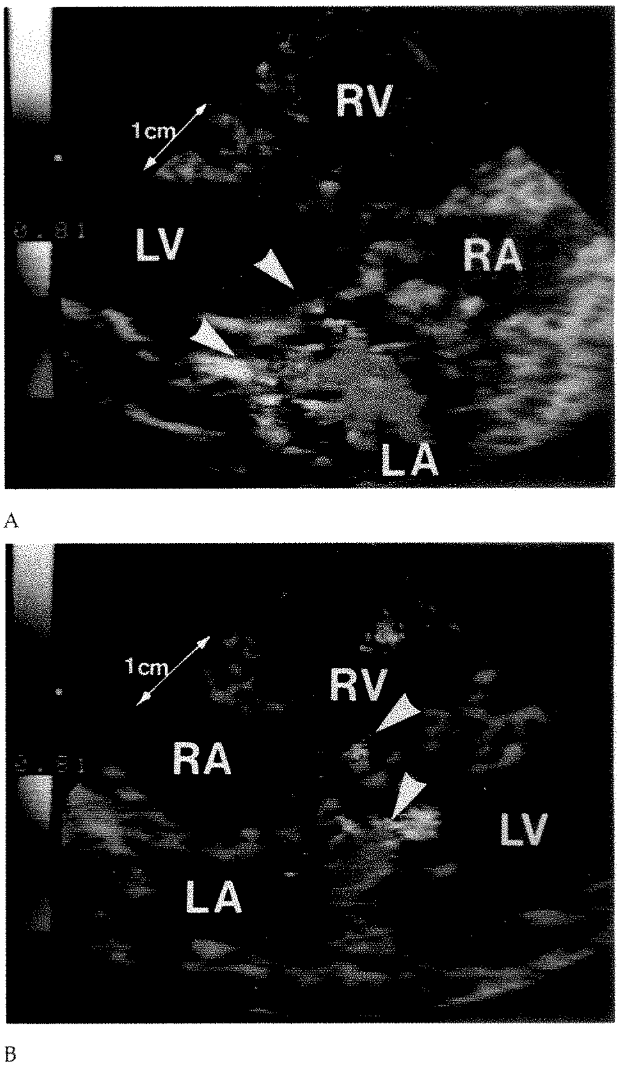

Fig 1. Intraoperative color flow mapping echocardiograms. (A) The transducer was positioned over the right ventricular outflow tract. Color flow mapping showed a jet of left atrioventricular valve regurgitation extending into the posterior portion of the left atrium consistent with moderate regurgitation. The arrows *point to two separate leaks; along the anterior and posterior aspects of the valve. (B) View from the apical portion of the right ventricle. The* arrows *point to mild degrees of left and right atrioventricular valve regurgitation.* (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

Postoperative Care

After surgical repair, each patient had central venous, left atrial, and main pulmonary artery catheters placed for postoperative hemodynamic monitoring. These catheters remained in place until the patient was extubated. The mean left atrial and main pulmonary arterial pressures were recorded for analysis three to five days after operation. All pressures were obtained simultaneously with or within one hour of the first postoperative CFM echocardiogram. After discharge all patients received digoxin and diuretic agents routinely. No patient was discharged with signs or symptoms of congestive heart failure in the opinion of the attending cardiologist and cardiothoracic surgeon.

Methods of Analysis

Left AV valve regurgitation was graded by CFM echocardiography depending on the distance that the regurgitant jet extended into the left atrium. Previous reports [12, 13] had shown a good correlation with angiographic estimates of mitral regurgitation using this method. Mild regurgitation (Fig 1B) was defined as a jet extending less than half the distance from the valve to the posterior left atrial wall. Moderate regurgitation (Fig 1A) was defined as regurgitant jet extending into the posterior half of the left atrial cavity. Severe insufficiency was defined as a regurgitant jet extending to the posterior wall of the left atrium or into the pulmonary veins. The degree of insufficiency was determined as the maximal extent of the color flow regurgitant jet in either the parasternal long axis, subxiphoid long axis, or apical four-chamber view. Preoperative AV valve regurgitation was graded similarly, depending on the distance the regurgitant jet extended into the left or right atrium.

Angiographic estimates of preoperative AV valve regurgitation were graded mild, moderate, or severe corresponding to grades I, II, and III to IV of the classification of Okamura and colleagues [14]. Angiograms were interpreted by a different physician blinded to the results of the CFM echocardiograms.

Correlation of scoring of valve regurgitation between angiography and CFM echocardiography was evaluated with a weighted κ statistic [15]. A nonparametric Spearman rank correlation test was used to correlate an individual preoperative or postoperative variable with the other variables. Statistical significance was measured at $p < 0.05$.

Results

Preoperative Evaluation

With the exception of the 11-year-old patient, mean weight at the time of the operation was 6.1 ± 2.4 kg (± standard deviation) (range, 3.4 to 11.3 kg). Pulmonary vascular resistance on room air averaged 3.3 ± 1.4 Woods units (range, 1.5 to 6 Woods units). Five of the 19 patients (26%) had aberrancies of the AV valve apparatus. The surgeon noted intraoperatively that 3 of these 5 patients had a relative "deficiency" of valve tissue on the left side at the posterior inferior bridging leaflet with a relatively large mural leaflet. One patient had a double-orifice left AV valve component, and another patient with AV septal defect and double-outlet right ventricle had a single papillary muscle in the left ventricle with the AV valve unbalanced to the right ventricle.

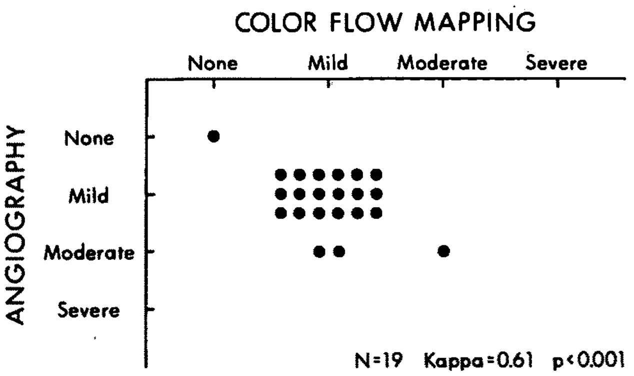

Fig 2. Magnitude of preoperative atrioventricular valvular regurgitation estimated by color flow mapping echocardiography compared with angiography.

Neither angiography nor CFM echocardiography demonstrated any instance of severe AV valve regurgitation (Fig 2). Angiography and CFM echocardiographic estimates of AV value regurgitation showed good agreement beyond chance ($\kappa = 0.61$, $p < 0.001$). Thus, our CFM echocardiographic estimates of preoperative AV valve regurgitation reflected angiographic findings.

Color Flow Mapping Echocardiography

Intraoperative CFM echocardiography after completion of the repair showed left AV valve regurgitation in 16 of 18 patients (89%), right AV valve regurgitation in 13 of 18 (72%), and residual ventricular septal defects in 4 of 18 (22%). Only 2 of 18 patients (17%) had no residual defects. All ventricular septal defects were discovered by abnormal left to right ventricular color flow jet only. In contrast, CFM echocardiography on postoperative days 3 to 5 showed left AV valve regurgitation in 17 of 19 (89%), right AV valve regurgitation in 15 of 19 (79%), small ventricular septal defects in 8 of 19 (42%), and no defects in 2 of 19 (11%). Thus, intraoperative CFM echocardiography showed a nearly perfect match with three- to five-day postoperative CFM echocardiography in demonstrating AV valve regurgitation but was relatively insensitive in detecting the small residual ventricular septal defects.

Intraoperative and early postoperative right AV valve regurgitation in each of the 19 cases was manifested by a narrow jet regurgitant directed toward the interatrial septum. In each case, the jet extended less than one half of the distance from the value of the distal right atrial wall and thus was considered mild regurgitation.

Only 3 of the 19 patients have not undergone late (more than 3 months) postoperative CFM echocardiographic studies. Of the 16 patients, 13 of 16 continue with left AV valve regurgitation and 8 of 16 continue with right AV valve regurgitation. However, no residual ventricular septal defects were noted as compared with the previous echocardiograms. Therefore, small ventricular septal defects detected by CFM echocardiography appeared to be

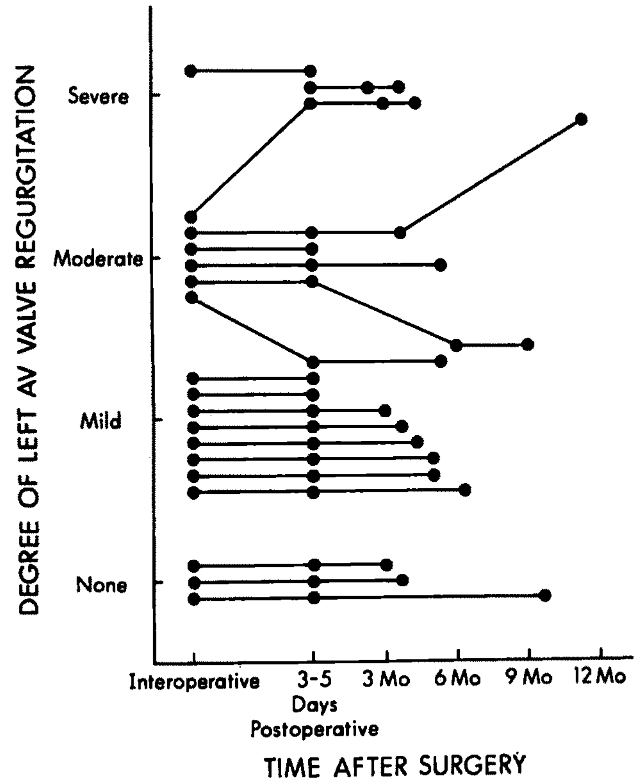

Fig 3. Estimate of left atrioventricular (AV) valvular regurgitation by color flow mapping echocardiography after AV septal defect repair and its change over time.

uniformly closed within 7 months of the operation, but AV valve regurgitation generally persisted.

Figure 3 demonstrates the CFM echocardiographic estimates of the degree of left AV valve regurgitation and its change with time. Excellent correlation ($r = 0.81$, $p < 0.001$) was obtained between estimates of intraoperative and three- to five-day postoperative left AV valve regurgitation by CFM echocardiography. Comparison of the magnitude of left AV valve regurgitation demonstrated continued excellent tracking on short-term follow-up. Thus, the intraoperative estimates of left AV valve regurgitation reliably reflected the later CFM echocardiographic findings.

Estimates of the degree of left AV valve regurgitation by intraoperative CFM echocardiography did not correlate with preoperative AV valve regurgitation ($r = 0.23$) or postoperative pulmonary artery pressures ($r = -0.05$). They correlated significantly with preoperative AV valve anatomical aberrancy (deficient valvar tissue, double-orifice left AV valve component, or single left ventricular papillary muscle) ($r = 0.58$, $p < 0.02$) and postoperative mean left atrial pressure ($r = 0.66$, $p < 0.003$).

Figure 4 shows the relationship between postoperative mean left atrial pressure and the CFM echocardiographic estimates of left AV valve regurgitation. The graph shows a definite trend toward increasing mean left atrial pressure with increasing severity of regurgitation. However, it also shows a large degree of overlap of the range of mean left atrial pressures for a given degree of valvular regurgitation. Thus, serious left AV valve insufficiency by CFM echocardiography could be associated with normal left atrial pressures.

Postoperative Course

All 19 patients survived initial repair of their AV septal defects. The patient who had severe left AV valve regurgitation intraoperatively (Fig 3) had signs of low cardiac output immediately after operation, could not be weaned from mechanical ventilatory support, and underwent successful left AV valvuloplasty seven days after his initial operation. This patient had a single left ventricular papillary muscle and still had mild left AV valve regurgitation and stenosis after his second operative procedure.

The other 18 patients were discharged from the hospital on digoxin and diuretics without evidence of congestive heart failure. Three of these 18 patients later developed congestive heart failure as manifested by tachypnea, poor exercise tolerance, growth failure, and increasing cardiomegaly on chest roentgenogram 3, 5, and 10 months after the initial AV septal defect repair. All 3 patients had had a deficiency of AV valvular tissue on the left side of their inferior bridging leaflets at the time of their initial operations. All 3 had severe left AV valve regurgitation by CFM echocardiography at the time congestive heart failure developed. Cardiac catheterization and angiography confirmed the presence of severe, 4+ [16] left AV valve regurgitation in these 3 patients. Their heart failure was not controlled with digoxin, diuretics, or vasodilators and

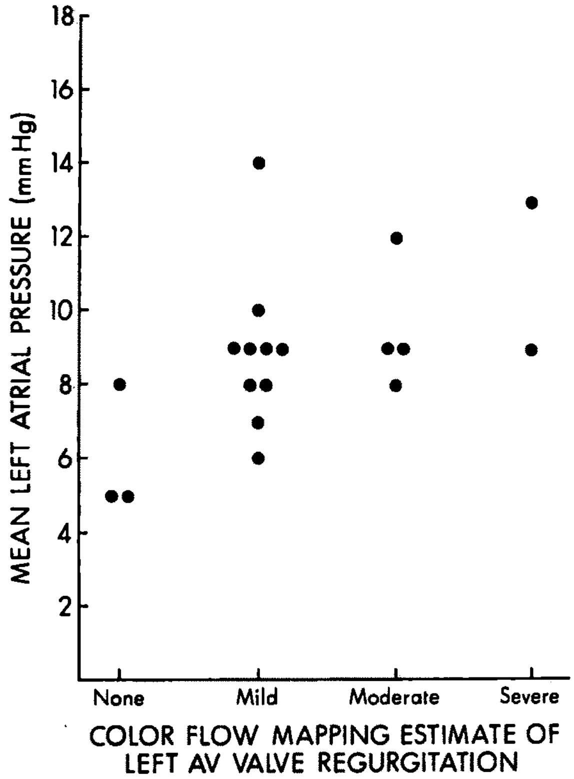

Fig 4. Relationship of mean left atrial pressure to color flow mapping estimate of left atrioventricular (AV) valve regurgitation three to five days after AV repair of septal defect.

Table 1. Relationship of Preoperative and Postoperative Variables to Development of Postoperative Congestive Heart Failure and Need for Reoperation

Variable	*r* Value	Significance[a]
Preoperative		
Age	−0.06	NS
Weight	−0.10	NS
Pulmonary resistance	0.13	NS
AV valve regurgitation	0.36	NS
Additional congenital heart defects	−0.15	NS
AV valve aberrancy	0.68	$p < 0.002$
Postoperative		
Mean pulmonary arterial pressure	−0.40	NS
Mean left atrial pressure	0.33	NS
Duration of hospital stay after repair	0.32	NS
Intraoperative CFM estimate of left AV valve regurgitation	0.68	$p < 0.002$
Three- to five-day postoperative estimate of left AV valve regurgitation	0.70	$p < 0.001$

[a] Significant at $p < 0.05$.

AV = atrioventricular; CFM = color flow mapping; NS = not significant.

required reoperation on the left AV valve. One of these 3 patients died shortly after left AV valve replacement.

Thus, in this series of consecutive patients with AV septal defect, the survival rate after AV septal defect repair was 100%, but in 4 of 19 patients (21%) postoperative congestive heart failure developed necessitating reoperation on the left AV valve. Table 1 shows that the need for reoperation correlated well with intraoperative and early postoperative CFM echocardiographic findings as well as with the preoperative presence of aberrant AV valve anatomy. Age, weight, additional congenital heart defects, preoperative pulmonary vascular resistance, preoperative AV valve regurgitation, postoperative length of hospital stay, and postoperative mean pulmonary arterial and left atrial pressure were not significantly correlated with eventual development of postoperative congestive heart failure and the need for reoperation on the left AV valve.

Comment

Preliminary studies have shown that CFM decreases the duration and improves the sensitivity of conventional echocardiographic and Doppler studies in the detection of residual defects after repair of congenital heart disease [8]. Thus, CFM echocardiography potentially could be a great help in open chest operative assessment of AV septal defect repair. The size of the color flow regurgitant jet is dependent on the velocity of flow more than the absolute regurgitant volume [17]; thus, jet dimensions are influenced by orifice size, changes in left ventricular afterload, time within systole, and regurgitant volume [17–19]. Furthermore, the measurement of color flow jet dimensions clearly is also influenced by the degree of color gain settings, transducer focal length, transducer angulation, brand of machine, and interobserver variability [20–23].

Despite these potential limitations, clinical studies in adults with mitral regurgitation have shown good correlation between angiography and CFM echocardiography in the quantitative assessment of the severity of mitral regurgitation [12, 13]. Intraoperative evaluation of mitral regurgitation in adults can agree with postoperative CFM echocardiographic and angiographic estimates of mitral regurgitation severity provided that intraoperative blood pressures are kept at preoperative levels [13].

In this study we documented that intraoperative CFM echocardiographic estimates of left AV valve regurgitation after surgical repair of AV septal defect correlated well with estimates obtained on short-term follow-up and with the development of congestive heart failure from postoperative left AV valve regurgitation. We also confirmed the findings of Meijboom and colleagues [24] that tissue deficiencies in the inferior bridging leaflet of the common AV valve can lead to severe postoperative left AV valve regurgitation.

From our study it appears that the single AV valve in an AV septal defect in infancy is relatively competent despite the anatomy of the valve leaflets. We, like McGrath and Gonzalez-Levin [6], found no correlation between the degree of preoperative AV valvular regurgitation and postoperative left AV valve regurgitation.

The use of the maximum length of regurgitant jet penetration into the left atrium in our study was similar to other studies assessing intraoperative and postoperative mitral insufficiency. Helmcke and co-workers [25] showed that the ratio of mitral regurgitant jet area to left atrial area more accurately correlates with the angiographic grade of mitral regurgitation than does measurement of maximal jet length. The length of time required to obtain and calculate this area ratio measurement might make it less useful in the intraoperative setting.

Transesophageal intraoperative CFM echocardiography is a better alternative than epicardial CFM echocardiography in adult patients in assessment of AV valvular regurgitation [26]. This technique could not be used in this study due to the relative size of the transesophageal transducer in relation to these patients. However, recent technological modifications [27] in the transesophageal transducer have produced a smaller transesophageal probe amenable to the size of the patients in this study.

Our study has demonstrated a very high prevalence of postoperative left and right AV valve regurgitation and residual ventricular septal defects observed by CFM echocardiography after repair of AV septal defects. These findings probably can be explained by the high sensitivity of CFM echocardiography in detecting intracardiac flows. Color flow mapping echocardiography is more sensitive than conventional pulsed Doppler echocardiography in the detection of multiple small ventricular septal defects [28]. The prevalence of left AV valve regurgitation in our series by CFM echocardiography was nearly 90%; the series of Chin and associates [2] showed a 65% prevalence of left AV valve regurgitation by postoperative angiography. Color flow mapping echocardiography has depicted mild degrees of AV valve regurgitation in a large percentage of normal adolescents and young adults without heart murmurs [29]. Preoperative CFM echocardiography in AV septal defects has detected mild degrees of left-sided and right-sided AV valve regurgitation not visualized with angiography [30]. Thus, the very high prevalence of mild postoperative left and right valve regurgitation in our series may also result from the high sensitivity of the CFM technique in demonstrating mild AV valve leaks.

In summary, intraoperative and postoperative CFM echocardiography is an accurate technique to predict which patients after repair of AV septal defects will have failure of the left AV valve and require reoperation. Postoperative pulmonary arterial or left atrial pressures were not helpful in predicting which patients would experience postoperative congestive heart failure. Clinical follow-up demonstrated that patients who had mild left AV valve regurgitation thrived and those with severe regurgitation did not. Therefore, early reoperation on the left AV valve after AV septal defect repair should be considered in any patient with severe left AV valve regurgitation demonstrated by CFM echocardiography. The timing of early reoperation and choice of procedure (valvuloplasty versus replacement) depend on the duration of the operative procedure and the clinical and hemodynamic status of the patient. Patients with moderate regurgitation warrant close follow-up for possible progression of left AV valve regurgitation.

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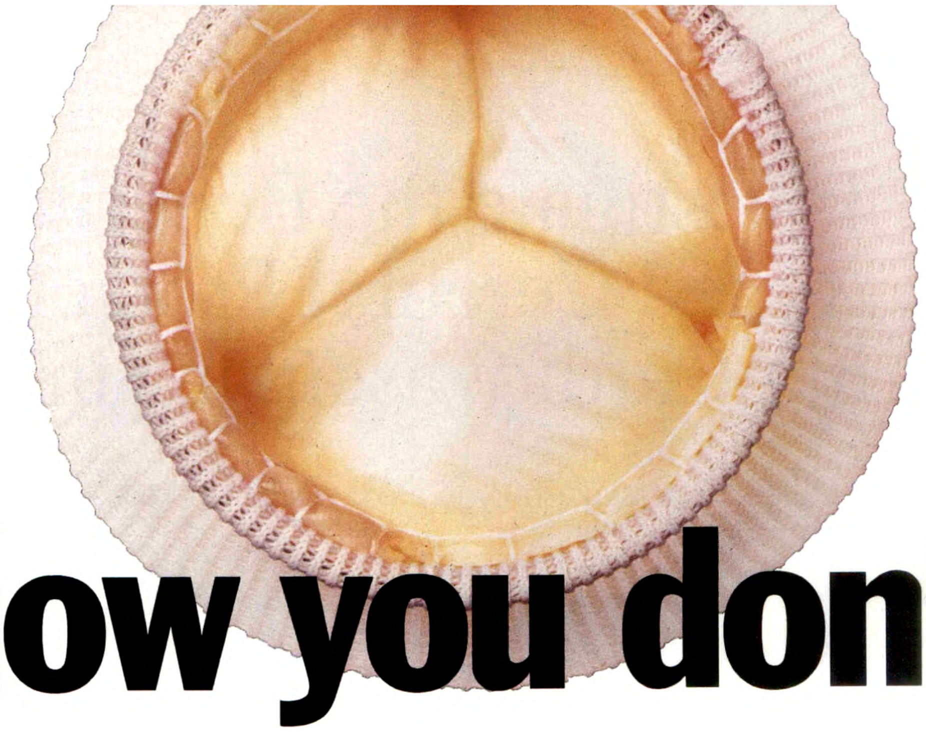

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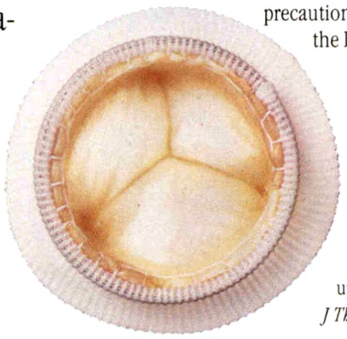

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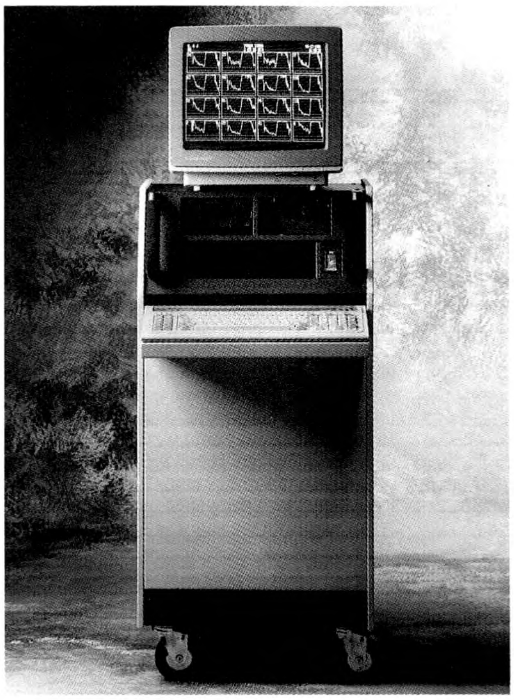

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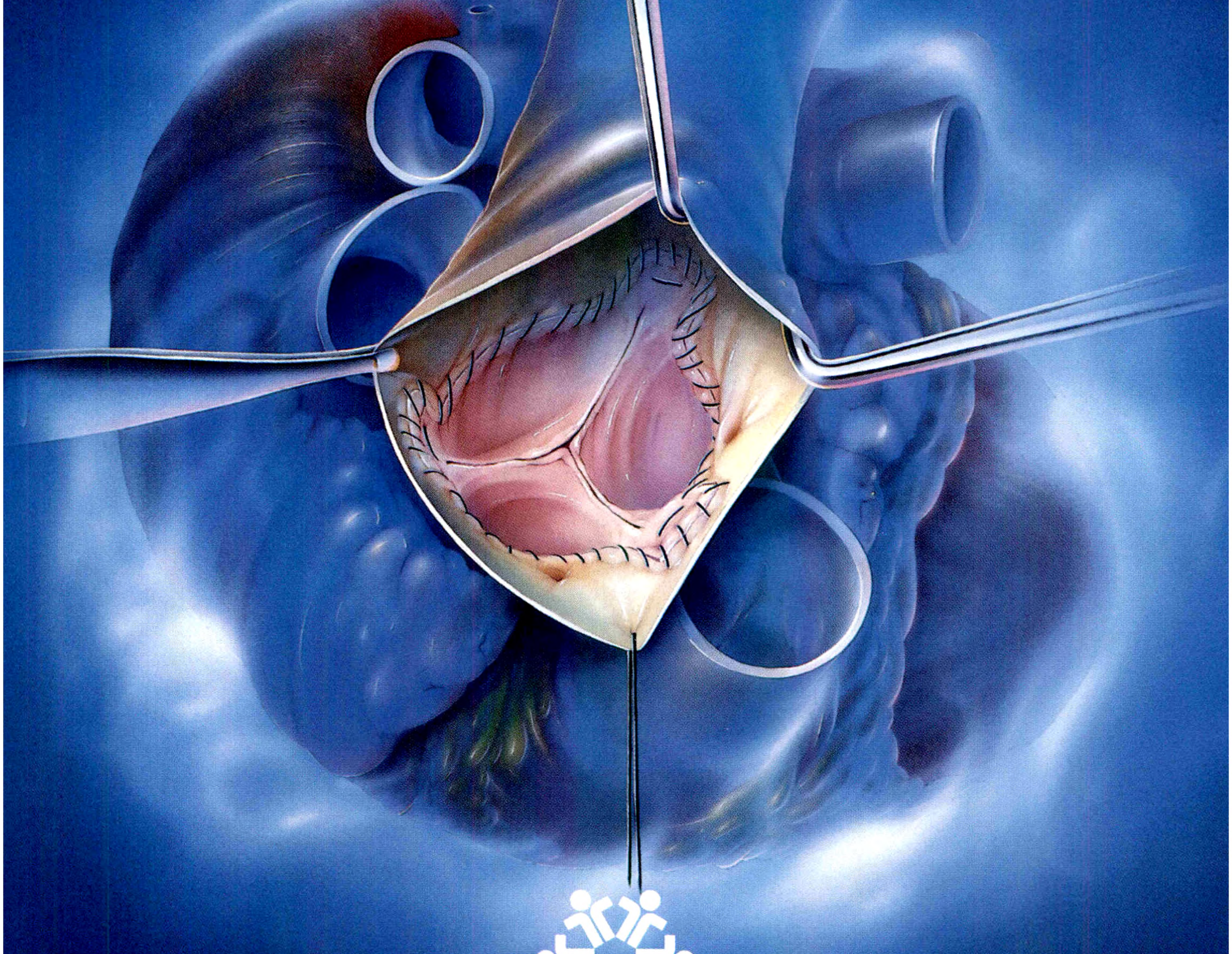

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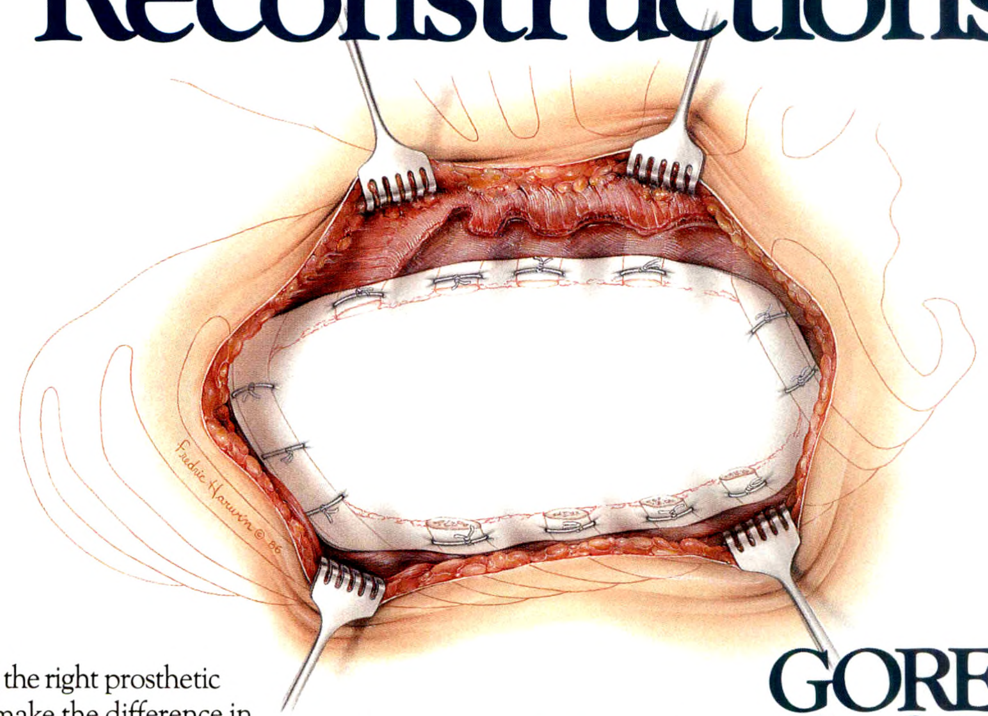

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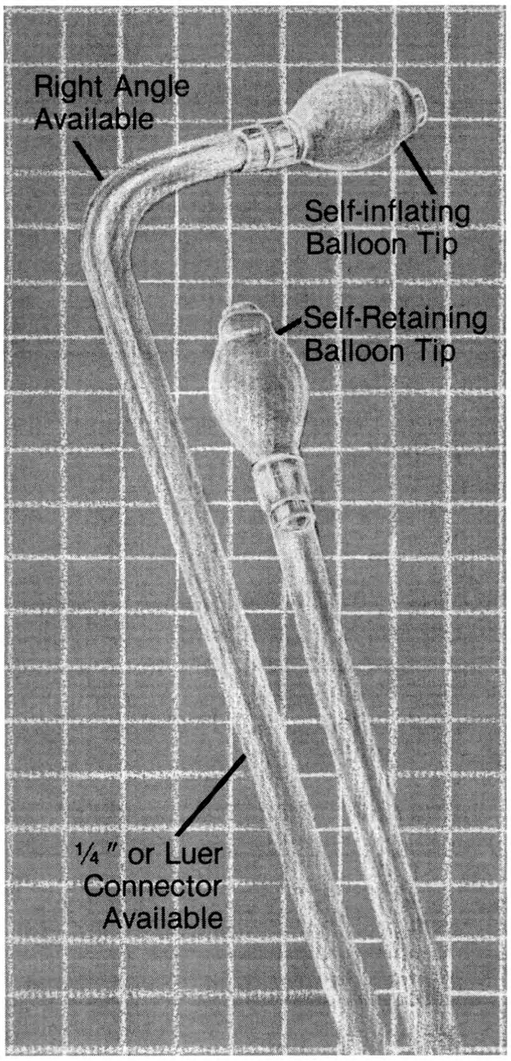
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INDICATIONS

Blenoxane should be considered a palliative treatment. It has been shown to be useful in the management of the following neoplasms either as a single agent or in proven combinations with other approved chemotherapeutic agents:

Squamous Cell Carcinoma—Head and neck (including mouth, tongue, tonsil, nasopharynx, oropharynx, sinus, palate, lip, buccal mucosa, gingiva, epiglottis, larynx), skin, penis, cervix, and vulva. The response to Blenoxane is poorer in patients with head and neck cancer previously irradiated.

Lymphomas—Hodgkin's, reticulum cell sarcoma, lymphosarcoma.

Testicular Carcinoma—Embryonal cell, choriocarcinoma, and teratocarcinoma.

CONTRAINDICATIONS

Blenoxane is contraindicated in patients who have demonstrated a hypersensitive or an idiosyncratic reaction to it.

WARNINGS

Patients receiving Blenoxane must be observed carefully and frequently during and after therapy. It should be used with extreme caution in patients with significant impairment of renal function or compromised pulmonary function.

Pulmonary toxicities occur in 10% of treated patients. In approximately 1%, the nonspecific pneumonitis induced by Blenoxane progresses to pulmonary fibrosis, and death. Although this is age and dose related, the toxicity is unpredictable. Frequent roentgenograms are recommended.

Idiosyncratic reactions similar to anaphylaxis have been reported in 1% of lymphoma patients treated with Blenoxane. Since these usually occur after the first or second dose, careful monitoring is essential after these doses.

Renal or hepatic toxicity, beginning as a deterioration in renal or liver function tests, have been reported infrequently. These toxicities may occur, however, at any time after initiation of therapy.

Usage in Pregnancy: Safe use of Blenoxane in pregnant women has not been established.

ADVERSE REACTIONS

Pulmonary—This is potentially the most serious side effect, occurring in approximately 10% of treated patients. The most frequent presentation is pneumonitis occasionally progressing to pulmonary fibrosis. Approximately 1% of patients treated have died of pulmonary fibrosis. Pulmonary toxicity is both dose and age related, being more common in patients over 70 years of age and in those receiving over 400 units total dose. This toxicity, however, is unpredictable and has been seen occasionally in young patients receiving low doses.

Because of lack of specificity of the clinical syndrome, the identification of patients with pulmonary toxicity due to Blenoxane has been extremely difficult. The earliest symptom associated with Blenoxane pulmonary toxicity is dyspnea. The earliest sign is fine rales.

Radiographically, Blenoxane-induced pneumonitis produces nonspecific patchy opacities, usually of the lower lung fields. The most common changes in pulmonary function tests are a decrease in total lung volume and a decrease in vital capacity. However, these changes are not predictive of the development of pulmonary fibrosis.

The microscopic tissue changes due to Blenoxane toxicity include bronchiolar squamous metaplasia, reactive macrophages, atypical alveolar epithelial cells, fibrinous edema, and interstitial fibrosis. The acute stage may involve capillary changes and subsequent fibrinous exudation into alveoli producing a change similar to hyaline membrane formation and progressing to a diffuse interstitial fibrosis resembling the Hamman-Rich syndrome. These microscopic findings are nonspecific, e.g., similar changes are seen in radiation pneumonitis, pneumocystic pneumonitis.

To monitor the onset of pulmonary toxicity, roentgenograms of the chest should be taken every 1 to 2 weeks. If pulmonary changes are noted, treatment should be discontinued until it can be determined if they are drug related. Recent studies have suggested that sequential measurement of the pulmonary diffusion capacity for carbon monoxide (DL_{CO}) during treatment with Blenoxane may be an indicator of subclinical pulmonary toxicity. It is recommended that the DL_{CO} be monitored monthly if it is to be employed to detect pulmonary toxicities, and thus the drug should be discontinued when the DL_{CO} falls below 30 to 35% of the pretreatment value.

Because of bleomycin's sensitization of lung tissue, patients who have received bleomycin are at greater risk of developing pulmonary toxicity when oxygen is administered in surgery. While long exposure to very high oxygen concentrations is a known cause of lung damage, after bleomycin administration, lung damage can occur at lower concentrations than usually would be considered safe. Suggested preventive measures are:

(1) Maintain $FI\ O_2$ at concentrations approximating that of room air (25%) during surgery and the postoperative period.
(2) Monitor carefully fluid replacement, focusing more on colloid administration rather than crystalloid.

Sudden onset of an acute chest pain syndrome suggestive of pleuropericarditis has been rarely reported during Blenoxane infusions. Although each patient must be individually evaluated, further courses of Blenoxane do not appear to be contraindicated.

Idiosyncratic Reactions—In approximately 1% of the lymphoma patients treated with Blenoxane, an idiosyncratic reaction, similar to anaphylaxis clinically, has been reported. The reaction may be immediate or delayed for several hours, and usually occurs after the first or second dose. It consists of hypotension, mental confusion, fever, chills, and wheezing. Treatment is symptomatic including volume expansion, pressor agents, antihistamines, and corticosteroids.

Integument and Mucous Membranes—These are the most frequent side effects being reported in approximately 50% of treated patients. These consist of erythema, rash, striae, vesiculation, hyperpigmentation, and tenderness of the skin. Hyperkeratosis, nail changes, alopecia, pruritus, and stomatitis have also been reported. It was necessary to discontinue Blenoxane therapy in 2% of treated patients because of these toxicities.

Skin toxicity is a relatively late manifestation usually developing in the 2nd and 3rd week of treatment after 150 to 200 units of Blenoxane have been administered and appears to be related to the cumulative dose.

Other—Vascular toxicities coincident with the use of Blenoxane in combination with other antineoplastic agents have been reported rarely. The events are clinically heterogeneous and may include myocardial infarction, cerebrovascular accident, thrombotic microangiopathy (HUS) or cerebral arteritis. Various mechanisms have been proposed for these vascular complications. There are also reports of Raynaud's phenomenon occurring in patients treated with Blenoxane in combination with vinblastine with or without cisplatin or, in a few cases, with Blenoxane as a single agent. It is currently unknown if the cause of Raynaud's phenomenon in these cases is the disease, underlying vascular compromise, Blenoxane, vinblastine, hypomagnesemia, or a combination of any of these factors.

Fever, chills, and vomiting were frequently reported side ef
Anorexia and weight loss are common and may persist long afte
mination of this medication. Pain at tumor site, phlebitis, and
local reactions were reported infrequently.

DOSAGE

Because of the possibility of an anaphylactoid reaction, lymp
patients should be treated with 2 units or less for the first 2 d
If no acute reaction occurs, then the regular dosage schedule
be followed.

The following dose schedule is recommended: **Squamous cel**
cinoma, lymphosarcoma, reticulum cell sarcoma, testi
carcinoma—0.25 to 0.50 units/kg (10 to 20 units/m^2)
intravenously, intramuscularly, or subcutaneously weekly or
weekly.

Hodgkin's Disease—0.25 to 0.50 units/kg (10 to 20 units/m^2)
intravenously, intramuscularly, or subcutaneously weekly or
weekly. After a 50% response, a maintenance dose of 1
daily or 5 units weekly intravenously or intramuscularly s
be given.

Pulmonary toxicity of Blenoxane appears to be dose related
a striking increase when the total dose is over 400 units. Total
over 400 units should be given with great caution.

Note: When Blenoxane is used in combination with
antineoplastic agents, pulmonary toxicities may occur at
doses.

Improvement of Hodgkin's Disease and testicular tumors is p
and noted within two weeks. If no improvement is seen by this
improvement is unlikely. Squamous cell cancers respond
slowly, sometimes requiring as long as three weeks befor
improvement is noted.

ADMINISTRATION

Blenoxane may be given by the intramuscular, intravenous, or
cutaneous routes.

Intramuscular or Subcutaneous—Dissolve the contents
Blenoxane vial in 1 to 5 mL of Sterile Water for Injection, U
Sodium Chloride for Injection, U.S.P., 5% Dextrose Injection, U
or Bacteriostatic Water for Injection, U.S.P.

Intravenous—Dissolve the contents of the vial in 5 mL or m
a solution suitable for injection, e.g., physiologic saline or glu
and administer slowly over a period of ten minutes.

STABILITY

The sterile powder is stable under refrigeration (2°-8°C) and s
not be used after the expiration date is reached.

Blenoxane is stable for 24 hours at room temperature in S
Chloride or 5% Dextrose Solution.

Blenoxane is stable for 24 hours in 5% Dextrose containing h
100 units per mL or 1000 units per mL.

Procedures for proper handling and disposal of anticancer
should be considered. Several guidelines on this subject have
published.[1-7] There is no general agreement that all of the
dures recommended in the guidelines are necessary or approp

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SUPPLY

Each vial contains 15 units of Blenoxane as sterile bleomycin s
U.S.P.

NDC 0015-3010-20

Manufactured by Nippon Kayaku Co., Ltd.
Tokyo, Japan

U.S. Patent No. 3,6

Mead Johnson
ONCOLOGY PRODUCTS
A Bristol-Myers Company
Evansville, Indiana 47721

Thymectomy for Myasthenia Gravis: Recent Observations and Comparisons With Past Experience

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To our previous report on the results of thymectomy for myasthenia gravis in 249 patients operated on between 1954 and 1981, we add a current review of 84 patients treated between 1982 and 1987. All patients underwent a median sternotomy, although this was performed after a bilateral submammary skin incision in most of the 57 female patients. There were no operative deaths, but one late death occurred at 5 months. During a mean follow-up of 3.6 years, 67 patients (80%) benefited from operation with remission achieved in 30 (36%) and improvement noted in 37 (44%). Acetylcholine receptor site antibody was present in 43 patients, of whom 19 (44%) achieved remission in contrast to 9 (27%) of the 33 patients without antibody. Hyperplasia of the excised thymus in 38 patients was associated with remission in 20 (53%) in contrast to remission in 7 (20%) of the 35 patients whose glands were "normal" or atrophic. The best prognosis was found in the 23 patients who had both receptor site antibody and thymic hyperplasia, as remission occurred in 15 of them (65%) in contrast to only 6 (27%) of the 22 patients who had neither factor. Remission rates (remissions per 1,000 patient-months of follow-up) for the present series (84 patients), the previous group (249 patients), and the overall group (333 patients) are 9.95, 6.13, and 6.62, respectively.

(Ann Thorac Surg 1989;48:551–5)

Progress continues to be made in our understanding of the pathophysiology of myasthenia gravis, even though some important aspects of this complex autoimmune process remain to be clarified. The basic problem involves blockade, accelerated degradation, or complement-related damage to acetylcholine receptor (AChR) mediated by autoantibodies, resulting in impaired neuromuscular transmission and symptoms of weakness and fatigue [1]. Four methods of treatment are currently used: anticholinesterase drugs (Mestinon [pyridostigmine bromide], Prostigmin [neostigmine bromide]) to enhance neuromuscular transmission; immunosuppression (prednisone, azathioprine) to decrease the immune response; plasmapheresis to remove some of the circulating autoantibodies; and thymectomy to remove a major source of antibody production.

In recent years, thymectomy has gained increasing acceptance as the most effective treatment for achieving sustained improvement in most patients with myasthenia gravis. The present report reviews our recent 5-year experience with thymectomy, and some comparisons are made with our previously reported series [2]. Over the past 32 years, more than 1,000 patients with myasthenia gravis have been evaluated, 333 of whom underwent thymectomy.

Material and Methods

Patient Population

In the 5-year period January 1983 through December 1987, at least 220 patients with a confirmed diagnosis of myasthenia gravis were evaluated at the University of California at Los Angeles Medical Center. Eighty-four of them underwent thymectomy. There were 57 female and 27 male patients. The age distribution of the male patients was unusual in that nearly 50% were more than 50 years of age (Table 1) in contrast to 17 (26%) of 66 patients in our previous experience [2].

Among the 84 patients, coexistent thymoma was found in 11 (6 female, 5 male patients). Eight of the thymomas were invasive.

All but 7 patients were in class II or III preoperatively. There were 5 patients in class I (mild generalized, including ocular, involvement), 31 in class II (moderately severe generalized and bulbar involvement), 46 in class III (acute generalized and bulbar involvement), and 2 in class IV (chronic, marked generalized and bulbar involvement).

Current follow-up information by physician examination, questionnaire, or phone conversation with the patient was obtained on 78 patients (93%) with a mean follow-up of 3.6 years. The remaining 6 patients had been followed for 1 month to 3 years, and this information was included in the data presented.

Reference will be made to our previously reported results [2] for 249 patients, which is of interest for comparative purposes. However, no attempt was made to obtain current information about this prior group of patients.

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Table 1. Age and Sex Distribution of 84 Patients

Age at Operation (yr)	Male	Female	Total
0–20	1	9	10
21–30	2	15	17
31–40	7	14	21
41–50	4	13	17
51–70	13	6	19
Total	27	57	84

Surgical Considerations

The criteria by which patients were selected for operation have remained essentially the same over the years. The majority of patients had severe and incapacitating myasthenia gravis, which compromised their life-style or ability to work. In many instances, the symptoms were progressive despite increasing amounts of medication. Adverse side effects to medication and the suspicion of thymoma were additional indications for operation. Operation early in the course of the disease rather than later was usually recommended in preference to continued attempts at medical control, including initiation of steroids, on the presumption that arrest or reversal of the myasthenic process would be more likely. In some instances, referral for operation was delayed many months because of patient or physician reluctance to accept the recommendation for operation.

Although steroids can be quite effective in providing symptomatic improvement, it is our practice not to use them preoperatively unless the patient is already receiving them. There were 18 patients on a regimen of steroids prior to operation; this medication was continued postoperatively at the same or reduced dosage in 13, and was tapered and discontinued in 5. Plasmapheresis treatments were given to 3 patients (three to six exchanges over a 1-week to 4-week period) with very severe and rapidly progressive symptoms. The result in each instance was a dramatic improvement in condition as indicated by increased strength and a reduction in medication requirements. This allowed for a much safer perioperative course.

Median sternotomy remains the incision of choice and was used in each of our patients. A transverse submammary skin incision was cosmetically preferable for most female patients. The entire thymus including the cervical stalks was completely excised, although no attempt was made to include fat or lymphatic tissue lateral to the thymic cervical pedicle where the vagus and recurrent nerves and other structures can be injured. Because aberrant thymic tissue will be found occasionally in the mediastinum lateral to the gland, both pleural envelopes were opened to allow meticulous dissection of all contiguous fat and any suspicious tissue that might contain islands of aberrant thymus. Lymph nodes and lymphatic tissue can sometimes resemble thymic tissue, especially on the left side where the lateral margin of the gland is less easily separated from these tissues. We frequently do biopsies of such suspicious tissue and occasionally have found small islands of thymus, but usually this is lymphatic tissue and fat. Great care was taken to avoid injury to the phrenic nerves, a catastrophe in the patient with severe myasthenia.

Complete removal of a coexistent thymoma frequently required excision of contiguous pericardium and portions of lung, innominate vein, or superior vena cava to provide an adequate surgical margin. In such patients, postoperative irradiation was given to lessen the likelihood of a local recurrence.

Extubation was accomplished in the operating room for patients who had minimal or no ventilatory compromise preoperatively. Most of the remaining patients were extubated after two to three hours of observation in the recovery room or subsequently in the intensive care unit if weaning from the respirator was required. Prolonged intubation (several days) was rare.

The determination for extubation was the patient's ability to maintain adequate ventilation (acceptable arterial blood gases) for two to three hours while breathing unassisted. Two patients required reintubation and were subsequently weaned successfully from the ventilator. Tracheostomy was not necessary in the postoperative period in any patient.

Mestinon was usually given the morning of operation and resumed no sooner than 12 hours postoperatively, initially at one-half the previous dose because of the increased sensitivity of most patients to the medication. In patients with dysphagia due to oropharyngeal involvement, a small, soft nasogastric feeding tube was useful, although parenteral Mestinon is now available.

Results

A comparison of the preoperative status with the patient's current condition allowed placement of the patient into one of the following categories: remission—no symptoms and no medication; improved—less medication and better control of symptoms; same—improvement modest or equivocal; worse—more medication and symptoms unchanged or increased; or dead—operative or late death.

Thymectomy was followed by remission in 11 male patients (41%) and improvement in 12 (44%), so that 23 (85%) of the 27 male patients received benefit from operation (Table 2). In 3 patients (11%) there was no major improvement. There were no operative deaths. One patient (4%) died 5 months after operation of complications related to a recurrent myasthenic crisis. Of the 57 female patients, 19 (33%) achieved remission, 25 (44%) had improvement, 12 (21%) remained the same, and 1 (2%) was worse. There were no deaths. Thus, 67 (80%) of the 84 patients benefited from operation.

It is of interest to compare these results with those in our previous experience [2] with 249 patients who had a more typical patient mix and a mean follow-up of 7.5 years, which is twice that of the present series (Table 3). There was the more typical 3:1 female to male ratio, and the female patients were more likely to benefit from operation than the male patients (91% versus 76% in the

Table 2. Results of Thymectomy for Myasthenia Gravis in 84 Patients (1982–1987)[a,b]

Postop Status	Male	Female	Total
Remission	11 (41)	19 (33)	30 (36)
Improved	12 (44)	25 (44)	37 (44)
Benefited from operation	23 (85)	44 (77)	67 (80)
Same	3	12	15
Worse	0	1	1
Dead	1[c]	0	1
Total	27	57	84

[a] Numbers in parentheses are percentages. [b] The mean follow-up was 3.6 years. [c] There was 1 late death at 5 months postoperatively.

previous report compared with 77% versus 85% in the present series). The overall benefit from operation in the two series was similar (87% in the previous report and 80% in this study).

Factors Affecting Results

Some of the factors that can influence the surgical results were evaluated.

INFLUENCE OF ACETYLCHOLINE RECEPTOR ANTIBODY. Serum for AChR antibodies was obtained from 76 of the 84 patients in the recent series (Table 4). Antibodies were detected in 43 patients, of whom 19 (44%) achieved remission and 21 had improvement; hence, 40 patients (93%) benefited from operation. In contrast, only 21 (64%) of the 33 patients in whom no antibody was detectable benefited from operation. Nine (27%) achieved remission, and 12 showed improvement.

INFLUENCE OF THYMUS PATHOLOGY. The presence of hyperplasia in the excised thymus was noted in 38 patients, of whom 20 (53%) achieved remission and 13 had improvement; hence, 33 (87%) benefited from operation (Table 5). A "normal" gland or one without specific diagnostic changes, eg, atrophy or involution, was found in 35 patients and was associated with remission in 7 (20%) and improvement in 17, so that 24 (69%) benefited from operation.

Table 3. Results of Thymectomy for Myasthenia Gravis in 249 Patients (1954–1981)[a,b]

Postop Status	Male	Female	Total
Remission	24 (36)	102 (56)	126 (51)
Improved	26 (39)	64 (35)	90 (36)
Benefited from operation	50 (76)	166 (91)	216 (87)
Same	7	10	17
Worse	1	0	1
Dead	8[c]	7[c]	15
Total	66	183	249

[a] Numbers in parentheses are percentages. [b] The mean follow-up was 7.5 years. [c] This figure includes 2 operative deaths.

Table 4. Influence of Receptor Site Antibodies on Results of Thymectomy in 84 Patients[a]

Antibodies	No. of Patients	Remission	Improved	Benefited From Operation
Present	43	19 (44)	21	40 (93)
Absent	33	9 (27)	12	21 (64)
Unknown	8	2	4	6 (75)
Total	84	30	37	67 (80)

[a] Numbers in parentheses are percentages.

Benefit from thymectomy and removal of the thymoma occurred in 10 (91%) of the 11 patients with thymoma. Three patients (27%) had remission and 7, improvement.

COMBINED INFLUENCE OF ACETYLCHOLINE RECEPTOR ANTIBODIES AND THYMUS HISTOLOGY. The combination of hyperplasia of the thymus gland and the presence of AChR antibodies was a favorable prognosticator. Fifteen patients achieved remission (65%) and 7 had improvement; hence, 22 (96%) of the 23 patients benefited from operation (Table 6). In contrast, the 22 patients without AChR antibodies and without hyperplasia did not have as favorable a response; 14 (64%) benefited from operation, and only 6 (27%) had remission.

Of the 11 patients with thymoma, all 10 with AChR antibodies benefited from operation (100%), with 3 (30%) achieving remission.

INFLUENCE OF PREOPERATIVE CLASSIFICATION. All but 7 patients were in class II (31) or class III (46) preoperatively; no comment concerning the effect of operation on class I (5) or class IV (2) patients can be made (Table 7). Among the class II patients, 12 (39%) went into remission and 12 had improvement; thus, 24 (77%) of the 31 patients benefited from operation. The results among the 46 class III patients were similar with 17 going into remission (37%) and 18 having improvement; 35 (76%) of the 46 patients received benefit from operation.

Statistical Analysis of Data

The data were analyzed to determine if there was a positive association between complete remission only and

Table 5. Influence of Thymus Pathology on Results of Thymectomy in 84 Patients[a]

Histology	No. of Patients	Remission	Improved	Benefited From Operation
Hyperplasia	38	20 (53)	13	33 (87)
No diagnostic change	35	7 (20)	17	24 (69)
Thymoma	11	3 (27)	7	10 (91)
Total	84	30	37	67 (80)

[a] Numbers in parentheses are percentages.

Table 6. Influence of Both Thymus Pathology and Receptor Site Antibodies on Results of Thymectomy in 84 Patients[a]

Clinical Features	No. of Patients	Remission	Improved	Benefited From Operation
Hyperplasia	38	20	13	33 (87)
Hyperplasia and antibodies	23	15 (65)	7	22 (96)[b]
Hyperplasia and no antibodies	11	3	4	7 (64)
No diagnostic changes	35	7	17	24 (69)
No diagnostic changes and antibodies	10	1	8	9 (90)
No diagnostic changes and no antibodies	22	6 (27)	8	14 (64)
Thymoma	11	3	7	10 (91)
Thymoma and antibodies	10	3	7	10 (100)

[a] Numbers in parentheses are percentages. [b] The difference between this category and that of hyperplasia and no antibodies is significant ($p < 0.05$).

histology of the thymus, presence of receptor site antibody, or functional class. Remission was calculated on the basis of rate per 1,000 patient-months, which helps adjust for the differing amount of follow-up. Because of the relatively small number of patients with complete data (76 of the 84 patients), a multivariate analysis of these three factors and others such as age and sex is not feasible.

Virtually all of the patients were in either class II or III, and the remission rate in these two categories was similar. The inadequate number of patients in classes I and IV in this series precludes any further comment.

A favorable trend in remission was observed in those patients with thymic hyperplasia and no antibodies as well as those with antibodies but without hyperplasia. However, only in those patients with the combination of thymic hyperplasia and antibodies present did the remission rate of 19.33/1,000 patient-months of follow-up reach significance with a p value of less than 0.05 (overall remission rate was 28 remissions/2,699 months of follow-up or 10.37 remissions/1,000 patient-months with a 95% confidence interval of 6.89 to 14.56).

Table 7. Influence of Preoperative Classification on Results of Thymectomy in 84 Patients

Class	No. of Patients	Postop Remission[a]
I	5	1
II	31	12 (39)
III	46	17 (37)
IV	2	0
Total	84	30 (36)

[a] Numbers in parentheses are percentages.

Table 8. Remissions After Thymectomy for Myasthenia Gravis in 333 Patients[a]

Variable	1954–1981	1982–1987	Overall (1954–1987)
No. of patients	249	84	333
No. of remissions	126	30	156
Follow-up (mo)			
Total	20,562	3,015	23,577
Mean	82.6	35.9	70.8
Remission rate[b]	6.13	9.95[c]	6.62

[a] Mean follow-up was 5.9 years. [b] This is remissions per 1,000 patient-months. [c] This is significantly different ($p < 0.05$) from the rate for the earlier group.

Attempts to compare the results from an earlier series with this experience are difficult because of subtle yet perhaps important differences in patient mix, surgical technique, case selection, and duration of follow-up. Recognizing these potential limitations, we compared the present series of 84 patients with our previously reported group of 249 patients and the overall group in regard to number of complete remissions and remission rate, as the duration of follow-up of the two series was different (Table 8). Remission rates were calculated on the basis of remissions per 1,000 patient-months of follow-up.

In the original group of 249 patients, 126 (51%) had remission within a total of 20,562 patient-months of follow-up (mean, 82.6 months). In the present series of 84 patients, 30 (36%) went into remission during a total of 3,015 patient-months of follow-up (mean follow-up, 35.9 months). In the overall group of 333 patients, 156 (47%) had remission during a total of 23,577 patient-months of follow-up (mean follow-up, 70.8 months). The remission rates for the three groups were 6.13, 9.95, and 6.62, respectively.

Comment

Clinical experience with large numbers of patients with myasthenia gravis treated with a variety of methods including steroids, immunosuppressive drugs, plasmapheresis, and thymectomy has been previously reviewed elsewhere [3, 4]. It is now generally accepted that removal of the thymus gland is the preferred treatment for most of these patients. There remains a difference of opinion as to the surgical approach that should be used and the extent to which tissues adjacent to the thymus that may contain aberrant thymic tissue should be excised.

Those who advocate the cervical approach [5, 6] do so because of their conviction that it is associated with less morbidity and a shorter hospital stay than a sternotomy, and that the results after operation are just as good as the results achieved by the more standard sternotomy route. At the other end of the spectrum are those [7] who are convinced that a much more aggressive approach is necessary to remove not only the obvious thymus but also all fat and other suspicious tissue in the neck and mediastinum that might harbor aberrant thymic tissue. This requires an additional cervical incision as well as the conventional median sternotomy, but such a "maximal" thymectomy is thought to be justified by the better results achieved compared with those achieved with lesser procedures.

We share the objective of removing all of the thymic tissue, and believe this can be most safely and consistently accomplished by using a median sternotomy approach. For reasons previously mentioned, it is considered imprudent to radically explore and excise contiguous tissues high in the mediastinum and in the lateral neck, as the yield of aberrant thymic tissue is low, and in our opinion, it has not been established that our results are inferior to those of the advocates of the more radical procedure.

Two observations are of interest in our most recent series, and they relate to the patient's response to thymectomy as influenced by AChR antibodies and histology of the thymus gland. Although antibodies were detected in only 51% of the patients (somewhat less than the usual 75% to 85% [8]), the results in this group compared with those in the group without antibodies reflected a favorable trend (remission, 44% versus 27%, and benefit from operation, 93% versus 64%). This difference was not statistically significant, and in the experience of Jaretzki and colleagues [7], there was no obvious relationship between AChR antibodies and the patient's response to thymectomy.

Of considerable interest was the response to operation of the 11 patients with thymoma, eight of which tumors were locally invasive but could be grossly excised. Past experience of most investigators indicates that the presence of a thymoma decreases the probability that thymectomy and removal of the thymoma will help the patient's myasthenic symptoms. This is most certainly true for the very invasive and extensive tumors. Of the 11 patients, 10 had receptor site antibody and each had an improvement in myasthenic symptoms including complete remission in 3. The presence of antibody in all but 1 of the patients with thymoma represents an unusual cluster of such patients. This may explain the better than expected operative benefit obtained, although the 27% remission rate is consistent with our past experience.

A favorable trend in remission rate was also noted in patients with thymic hyperplasia compared with those whose gland appeared normal or atrophic. However, no statistically significant difference between these groups could be demonstrated.

The best prognosticator of a favorable result following thymectomy was the presence of both thymic hyperplasia and receptor site antibodies. When this group was compared with patients with neither antibodies nor hyperplasia, a significantly different remission rate ($p < 0.05$) was demonstrated (19.33/1,000 patient-months of follow-up versus 6.98, and total remissions, 65% versus 27%).

The percentage of patients who have undergone remission in the present series is less than in our initial group (51% versus 36%). This was unexpected because in recent years, our thymectomy procedure has become somewhat more extensive in the removal of contiguous mediastinal fat and other possible thymus-containing tissue. However, when the data are compared on the basis of duration of patient-months of follow-up, the remission rate of 9.95/1,000 patient-months of follow-up in the recent series is significantly greater ($p < 0.05$) than the 6.13 obtained in our previous group of patients. This suggests that over a comparable follow-up, the eventual percentage of remissions in the present series will exceed that observed in the initial group. What factors are responsible for this difference, eg, differing patient populations or extent of the surgical procedure, will be difficult to identify with certainty.

The value of thymectomy in improving the symptoms of patients with myasthenia gravis is established. The search continues for better markers to select the optimal candidates for operation, as well as to determine how aggressive the surgeon should be in attempting to remove all conceivable foci of thymic tissue.

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Use of Artificial Intelligence for the Preoperative Diagnosis of Pulmonary Lesions

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The relatively new field of artificial intelligence has spawned a variety of techniques associated with computer-assisted diagnosis. These techniques have been applied to the diagnosis of pulmonary lesions, but previous reports have focused on medical rather than surgical populations and the results have been evaluated using only retrospective patient surveys. We used a Bayesian algorithm to develop a diagnostic computer model for prospectively evaluating patients undergoing thoracotomy for suspected pulmonary malignancy. Patients who had a preoperative diagnosis were not included. Preoperative clinical and radiographic parameters for 100 consecutive patients were prospectively entered into the diagnostic model, which then categorized the lesion as benign or malignant. The computer predictions agreed with the final histological diagnosis in 95 of the 100 patients. The sensitivity was 96% and the specificity was 89% for this prospective series. These results indicate that the computer-assisted diagnosis of pulmonary lesions may have a role in this clinical setting.

(Ann Thorac Surg 1989;48:556–9)

There are well-accepted clinical and radiographic parameters associated with malignancy, but the diagnosis of new pulmonary lesions is nevertheless quite challenging. Because of this difficulty in making an accurate preoperative diagnosis of such lesions, patients may undergo unnecessary invasive procedures and may encounter some delay before receiving treatment.

Recently, artificial intelligence techniques have been applied to this problem [1–4]. Most approaches deal with computer-assisted diagnosis based on expert systems or Bayesian theory, and several studies have reported accurate results [1–4]. The goal of these studies is to provide an accurate diagnostic adjunct that can be integrated with more conventional information to arrive at a clinical diagnosis.

We have used a Bayesian model to predict the diagnosis of newly discovered pulmonary lesions. Our initial experience involved the prospective evaluation of patients undergoing thoracotomy for suspected bronchogenic cancer. The model produced a 96% diagnostic accuracy, but more than one third of these patients had a histological diagnosis before thoracotomy, indicating that the model held promise for practical clinical application. The true test, however, must address the diagnosis of patients in whom the preoperative diagnosis is not known. The purpose of the present study was to use computer-assisted diagnosis to categorize pulmonary lesions as being benign or malignant in a population of patients in whom the preoperative diagnosis had not been established.

Theory

We previously described a step-by-step approach [5] that serves as a technical guide for physicians and other individuals who wish to develop Bayesian studies. The more intricate details of Bayesian theory are covered in that reference, but a general overview of this approach may be helpful to introduce our present work.

Bayesian algorithms use clinical observations of previously evaluated patients to predict the diagnosis of new patients. These clinical observations usually take the form of risk factors that are selected for their ability to discriminate between diagnostic categories. The frequency with which the risk factors are found in each diagnostic category makes up a conditional probability matrix (CPM) that is incorporated into a computerized Bayesian algorithm.

The conditional probabilities are derived from retrospective patient surveys, published data, or physician estimates [1, 2, 5, 6]. The technique used to generate the CPM determines the clinical data base on which diagnostic predictions are made. For that reason, a retrospective patient survey from one's own institution ensures that the model is tailored to reflect the unique experience of that institution.

After the model has been developed, a new patient can be evaluated by tabulating the presence or absence of each risk factor for that patient. The Bayesian algorithm then uses the clinical experience embodied in the CPM to

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calculate the probability that the patient will fall into a given diagnostic category.

Material and Methods

In this study, two diagnostic categories were considered: "benign" and "malignant." The risk factors (Table 1) were restricted to clinical and radiographic parameters that are readily available as part of the routine preoperative evaluation for patients suspected of having pulmonary malignancy. We selected the factors that we considered important in discriminating between benign and malignant lesions [1, 4, 7]. The associated conditional probabilities were derived from a combination of physician estimates and a retrospective review of our clinical experience. This information was used to develop a computerized Bayesian algorithm that applied the CPM to predict the diagnosis of new patients.

The patient population for entry into this model was drawn from the recent operative experience at our institution. From January 1986 to January 1988, 165 consecutive patients underwent thoracotomy for suspected pulmonary malignancy. All patients underwent our usual preoperative diagnostic evaluation. Chest roentgenograms were obtained for each patient, and most had computed tomography of the chest.

Each patient underwent fiberoptic bronchoscopy and, if an endobronchial lesion was identified, a biopsy specimen was taken. Otherwise, brushings and washings of appropriate areas were performed. Generally, an attempt was made to perform a transthoracic needle biopsy if the lesion was in a peripheral location. Patients with lesions believed to be accessible by transbronchial biopsy also underwent that procedure. If computed tomography identified mediastinal lymph nodes larger than 1.5 cm in diameter, a staging procedure with cervical mediastinoscopy or anterior mediastinotomy was performed.

Using this approach, we confirmed a preoperative diagnosis of cancer in 65 patients. A preoperative diagnosis could not be established in the remaining 100 patients, who constituted the study population. A posterolateral thoracotomy was performed on each patient, and the pulmonary lesion was completely excised and submitted for histological examination.

The presence or absence of the risk factors (Table 1) was entered into the model for each patient, and the Bayesian algorithm then calculated the probability that the lesion was benign or malignant. The "diagnostic prediction" for a given patient was the alternative (benign or malignant) calculated to have the higher probability. To test the validity of the diagnostic model, this calculated result was compared with the final histological diagnosis obtained from the excised specimen.

Results

Table 1 shows a clinical profile of the patient population. Of the 100 patients undergoing thoracotomy, 82 had malignant lesions and the remaining 18 had benign lesions. The model correctly categorized the lesion as benign or malignant in 95 of the 100 cases, yielding a 95%

Table 1. Preoperative Patient Characteristics

No. of Patients	Total (n = 100)	Cancer[a] (n = 82)	Benign[a] (n = 18)
Age < 45 yr	16	10 (12)	6 (33)
Age ≥ 45 yr	84	72 (88)	12 (67)
Male	71	56 (68)	15 (83)
Female	29	26 (32)	3 (17)
Smoking history	81	69 (84)	12 (67)
Weight loss	13	13 (16)	0 (0)
Hemoptysis	11	10 (12)	1 (6)
New cough	33	31 (38)	2 (11)
Chest pain	7	6 (7)	1 (6)
HPO symptoms	3	3 (4)	0 (0)
Bloody effusion	0	0 (0)	0 (0)
Previous cancer	25	24 (29)	1 (6)
Lesion < 3 cm	47	32 (39)	15 (83)
Lesion 3.1–5 cm	41	39 (48)	2 (11)
Lesion > 6 cm	12	11 (13)	1 (6)
Cavitation (thick wall)	8	7 (9)	1 (6)
Cavitation (thin wall)	1	0 (0)	1 (6)
Smooth margins	23	8 (10)	15 (83)
Slightly irregular margins	42	41 (50)	1 (6)
Very irregular margins	29	28 (34)	1 (6)
Lobulation	10	9 (11)	1 (6)
Spiculation	15	15 (18)	0 (0)
Homogeneous	70	57 (70)	13 (72)
Inhomogeneous	26	23 (28)	3 (17)
Tracheal deviation	3	3 (4)	0 (0)
No calcification	95	78 (95)	17 (94)
Eccentric calcification	0	0 (0)	0 (0)
Central/popcorn calcification	0	0 (0)	0 (0)
Atelectasis	20	19 (23)	1 (6)
Chest wall abutment	16	14 (17)	2 (11)
Chest wall invasion	2	2 (2)	0 (0)
Mediastinal abutment	14	13 (16)	1 (6)
N1 enlargement	9	9 (11)	0 (0)
N2 enlargement	11	11 (13)	0 (0)
No size increase in 6 mo	4	1 (1)	3 (17)
Size increase in 6 mo	49	44 (54)	5 (28)
Effusion on chest film	3	3 (4)	0 (0)
Middle lobe lesion	5	2 (2)	3 (17)
Bilateral lesions	9	8 (10)	1 (6)
Multiple unilateral lesions	5	4 (5)	1 (6)
No lesions on FOB	69	52 (63)	17 (94)
Endobronchial lesion seen	4	4 (5)	0 (0)
Extrinsic compression on FOB	2	2 (2)	0 (0)
Blunted carina on FOB	0	0 (0)	0 (0)

[a] Numbers in parentheses are percentages.

FOB = fiberoptic bronchoscopy; HPO = hypertrophic pulmonary osteoarthropathy.

Table 2. Confidence Limits of Selected Indexes

Index	90% Confidence Level (%)	70% Confidence Level (%)
Accuracy (95%)	89–98	92–97
Sensitivity (96%)	90–99	93–98
Specificity (89%)	68–98	76–96
Predictive value of		
Positive test (97%)	92–99	94–99
Negative test (84%)	63–95	71–93

accuracy. Sixteen of the 18 benign lesions and 79 of the 82 malignant lesions were correctly assigned, thereby producing two false-positive and three false-negative results. This yielded a sensitivity of 96% and a specificity of 89% with confidence limits as shown in Table 2.

The predictive value of a positive test is the probability that a patient with a positive test does in fact have the disease in question [8]. In the present context, a positive test refers to the Bayesian prediction of cancer and the disease in question is pulmonary malignancy. The predictive value of a positive test was 97% in this series. The predictive value of a negative test [8], ie, the probability that a patient with a benign test result has a benign pulmonary lesion, was 84% for this model.

The likelihood ratio [8] is a useful entity that provides an intuitive measure of test accuracy. In this study, the likelihood ratio for a positive test was 8.7, which indicates that a person with cancer is 8.7 times more likely to test positive than a person with benign disease. Conversely, the likelihood ratio for a negative test is 0.04, indicating that a person with cancer is 0.04 times as likely to test negative as compared with a person with a benign process.

The results discussed apply to a discrete "yes or no" application of the diagnostic algorithm. Actually, the model calculates the probability of each diagnostic alternative. We arbitrarily selected the alternative with the higher probability as the computer diagnosis, but examination of a breakdown of the actual calculated probabilities for these patients is useful.

Table 3 shows the results for calculating the probability of a benign lesion. There was reasonable agreement between the observed and predicted results, but the most salient feature of Table 3 concerns the patients predicted to have a very low probability of benign disease. The model predicted that the probability of having a benign lesion was less than 5% for 57 patients. In fact, none of these patients had benign disease. Conversely, all 57 patients with greater than 95% probability of having a malignant lesion were found to have cancer at the time of operation.

Comment

Several studies have reported the use of computerized mathematical algorithms to assist in the diagnosis of pulmonary lesions. As the field of computer-assisted diagnosis has evolved, it has become apparent that Bayesian theory is most suited to this task; in fact, all successful applications have used this technique [1, 4–6, 9]. Earlier reports examined the entire spectrum of patients with pulmonary lesions, but we focused on a more select and more challenging subgroup. By restricting our analysis to patients undergoing operation without a preoperative diagnosis, we directly addressed the most compelling practical problem in this clinical context.

Surprisingly, none of the previous studies used a prospective analysis to validate the diagnostic model. Instead, the test group was the same population of patients that was used to generate the model [1, 2, 9]. Clearly, the goal of diagnostic mathematical algorithms should be to predict the diagnosis of new patients rather than to model previously evaluated patients. In the present study, the Bayesian algorithm was developed before our prospective test group was examined so that the evaluation of these 100 prospective patients was completely independent of the patient population used to derive the model.

The model we present has proved to be an accurate diagnostic tool that other researchers may find helpful. However, a program developed in one institution may not be directly applicable to other institutions. The way in which the CPM is developed will effectively tailor the model to reflect the clinical experience of that institution. This is both an advantage and a disadvantage to potential users. The disadvantage is that a program developed in one practice cannot usually be used in another practice because of the differences in patient population and differences in the approach to preoperative evaluation. For example, a group that routinely performs mediastinoscopy on all patients will not have the same mix of patients undergoing thoracotomy as we do, nor will those groups that only rarely use mediastinoscopy. The advantage is that one can create a diagnostic model that conforms closely to the unique approach that is used in a given institution. Most surgeons are aware of the difficulties inherent in extrapolating information from reported series for use in their own practice. Problems of this kind can be obviated by the ability to tailor the model to reflect the specific population of any given hospital.

Table 3. Predicted Versus Observed Results for Benign Lesions

Predicted Probability of Benign Lesions (%)	Observed Frequency of Benign Lesions[a]
<5	0% (0/57)
5–25	7% (1/15)
25–50	11% (1/9)
50–75	67% (4/6)
>75	92% (12/13)

[a] In parentheses, the denominator is the number of patients with the predicted probability of benign disease that lies within the range shown in the first column. The numerator is the number of patients who actually had a benign lesion (eg, 13 patients were predicted to have >75% probability of benign disease and 12 of those patients did have a benign lesion).

The risk factors for malignancy (Table 1) were selected from our own clinical observations and from reports in the literature [1, 2, 4, 7]. We do not contend that these parameters are the only ones of importance, nor do we contend that all of them are necessary in such an analysis. One of the advantages of Bayesian theory is that one does not pay a penalty for selecting a risk factor that has little impact on the diagnosis. If a parameter does not significantly discriminate between diagnostic categories, the derived conditional probabilities for that factor will be approximately equal for each diagnosis, so that the factor simply has little mathematical consequence in the final calculations [5, 6, 9]. Because of this, we have been liberal in selecting our preoperative clinical and radiographic parameters, and we encourage other researchers to adopt a similar philosophy. The choice of these factors is discussed in some detail in other reports [3–6], which may be useful to potential investigators.

We have shown that computer-assisted diagnosis can provide accurate results in the preoperative assessment of pulmonary lesions, but the question of how to use this information is still unsettled. Certainly any test with more than 90% accuracy should be welcome in this clinical setting, particularly if the test is not invasive and has no morbidity or cost.

The utility of our approach is supported by the fact that the sensitivities, specificities, and predictive accuracy of the results were high both in the present study and in our previous work [4], even though the test populations were different and the sample populations were only partially overlapping.

This type of test does not dictate therapy [3–5, 9], but rather serves as an adjunct that should be evaluated with other preoperative tests to arrive at a clinical diagnosis. We have not used the test to influence our preoperative management, but such consideration may be warranted. If the model predicts that the probability of cancer is greater than 95% and our clinical judgment is consistent with a malignant lesion, perhaps it would be appropriate to perform thoracotomy without additional procedures that might otherwise have been done. Under these circumstances, the test provides an objective, statistically rigorous basis for our approach. Even this application may appear radical to some investigators, but we often use other tests, such as transthoracic needle biopsy, that are more invasive, more morbid, more costly, and less accurate.

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Evolution of the Management of Anomalous Left Coronary Artery: A New Surgical Approach

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Twelve patients with anomalous left coronary artery (LCA) from the pulmonary artery were treated surgically. Mean age was 13 months and mean weight, 7.7 kg. One of the following techniques was used in 8 patients: suture obliteration of the coronary orifice, ligation of the anomalous LCA and grafting of the internal mammary artery to the distal LCA, aortic reimplantation of the anomalous LCA, creation of an aortopulmonary window, and direct anastomosis to the right subclavian artery. In 4 patients, a new technique to establish a two-coronary artery system involved tube extension of the anomalous LCA with pulmonary artery wall and then anastomosis to the right subclavian artery using cardiopulmonary bypass without aortic cross-clamping. There were two hospital deaths (17%) and one late death. The condition of the 9 long-term survivors was improved at late follow-up. Postoperative assessment showed smaller Q wave on the electrocardiogram, reduced cardiac size, and good left ventricular function. All patients in whom the repair was done using the new technique were free from postoperative cardiac complications, had shortened hospital stays, and were alive with a patent two-coronary artery system at late follow-up. We believe this approach is a safer method for establishing a two-coronary artery system in infants and small children with an anomalous LCA arising from the pulmonary artery.

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Anomalous origin of the left coronary artery (LCA) from the pulmonary artery is a rare congenital cardiac malformation and, if left surgically untreated, carries a mortality rate of 80% to 90% within the first year of life [1–3]. Various surgical methods have been reported. However, it is generally accepted that establishment of a two-coronary artery system is desirable to reduce the number of early and late deaths [1, 4–6]. Coronary reimplantation [7, 8], aortocoronary bypass grafts with various materials [8–11], left subclavian–coronary artery anastomosis [12, 13], and creation of an aortopulmonary window with an intrapulmonary baffle using pericardium [14] or pulmonary artery tissue [15] have been proposed as methods for creating a two-coronary artery system. These techniques are difficult in infants, and in patients with an already compromised myocardium they provide suboptimal protection.

In this report, we present our experience with 12 patients with anomalous LCA arising from the pulmonary artery, and describe the evolution of a new, safer surgical technique to establish a two-coronary artery system.

Material and Methods

Patient Population

Between 1973 and 1987, 12 children underwent surgical treatment of anomalous LCA arising from the pulmonary artery at the University of Colorado Health Sciences Center and The Children's Hospital in Denver. There were 8 girls and 4 boys. All had the operation in infancy or early childhood (mean age, 13 months), and 1 patient required two operations. Body weight at the time of the initial operation ranged from 4.6 to 12.3 kg (mean weight, 7.7 kg).

Two patients were found to have anomalous LCA when evaluated for asymptomatic murmurs. The remaining 10 patients were seen with myocardial ischemia or cardiac failure, or both, with failure to thrive. Ischemic changes on the electrocardiogram (ECG) and cardiomegaly were present in all 12 patients. In every instance, cardiac catheterization confirmed the diagnosis preoperatively. Additional cardiovascular malformations were present in 7 patients (Table 1).

Technique

The operations were performed using cardiopulmonary bypass. Six different surgical procedures were used (see Table 1). Suture obliteration of the orifice of the anomalous LCA was performed in 2 patients. Another child underwent a similar procedure at the second operation, which followed failure of an internal mammary artery graft. Ligation of the anomalous LCA and pedicled internal mammary artery grafting to the distal left coronary artery was done in 1 patient. This patient required reoperation because of failure of the graft and recanalization of the anomalous LCA. Aortic reimplantation of the anomalous LCA was used in 2 patients. Redirection of aortic blood into the anomalous LCA by the creation of an aortopulmonary window was performed in 2 patients.

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Table 1. Preoperative and Operative Data

Patient No.	Age (mo)	Sex	Weight (kg)	Anomalous LCA Origin	Associated Lesions	Symptoms	Procedure
1	36	F	11.3	Main PA	None	MI, cardiac failure	Ligation of anomalous LCA, mammary artery graft
	72					Angina	Suture obliteration of anomalous LCA orifice
2	12	F	8.7	Main PA	None	Angina, cardiac failure	Aortic reimplantation
3	4	M	5.3	Main PA	PDA	MI, cardiac failure	Suture obliteration of anomalous LCA orifice
4	5	M	5.9	Main PA	MR	Cardiac failure	Aortic reimplantation
5	9	F	8.0	Main PA	None	Cardiac failure	Suture obliteration of anomalous LCA orifice
6	22	F	11.1	Main PA	None	Asymptomatic	Aortopulmonary window
7	11	F	4.6	Right PA	Aortic coarctation, VSD, hypoplastic left ventricle	Cardiac failure	RSA to anomalous LCA
8	40	M	12.3	Main PA	Aortic coarctation, PDA	MI	Aortopulmonary window
9	9	M	7.5	Main PA	MR	Cardiac failure	RSA to PA to anomalous LCA
10	3	F	5.4	Main PA	MR	Cardiac failure	RSA to PA to anomalous LCA
11	4	F	5.3	Main PA	None	Cardiac failure	RSA to PA to anomalous LCA
12	4	F	7.1	Main PA	ASD	Asymptomatic	RSA to PA to anomalous LCA

ASD = atrial septal defect; LCA = left coronary artery; MI = myocardial infarction; MR = mitral regurgitation; PA = pulmonary artery; PDA = patent ductus arteriosus; RSA = right subclavian artery; VSD = ventricular septal defect.

End-to-end anastomosis of the right subclavian artery to the anomalous LCA was performed in 1 patient.

In 4 patients, a new technique was used (Fig 1). The anomalous LCA was elongated using pulmonary artery wall (Fig 2) followed by end-to-end anastomosis to the right subclavian artery (Fig 3). The procedure was carried out with mild hypothermia on cardiopulmonary bypass but without aortic cross-clamping (Fig 1). An autologous pericardial patch was used to repair the pulmonary artery.

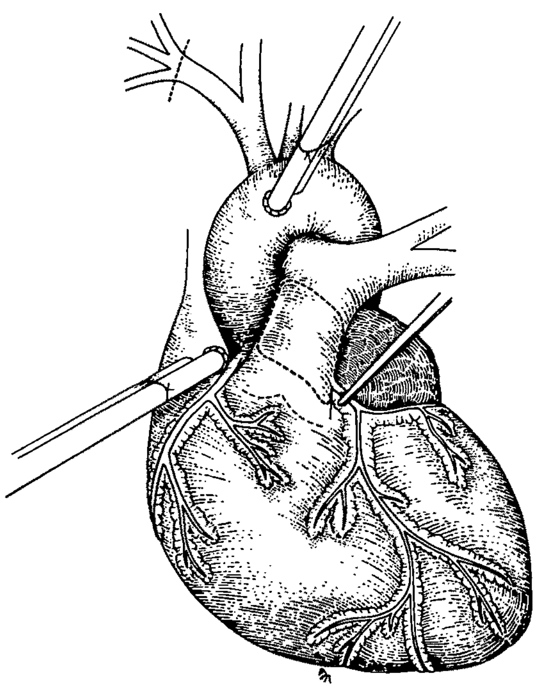

Fig 1. A new technique for repair of anomalous left coronary artery.

Results

Two patients (17%) could not be weaned from cardiopulmonary bypass. One of them underwent aortic reimplantation of the anomalous LCA, but bleeding secondary to anastomotic tension necessitated reinstitution of cardiopulmonary bypass, which resulted in further myocardial damage. The second patient had complex cardiac anomalies and underwent intraoperative balloon angioplasty of a recoarctation initially, and an end-to-end anastomosis of the anomalous LCA to the right subclavian artery. Autopsy confirmed the presence of a hypoplastic left ventricle and aorta, a ventricular septal defect, and a scarred interventricular septum.

There was one late death, which occurred in the first patient in our series. During the first operation, an attempt at saphenous vein grafting was unsuccessful. A pedicled internal mammary artery graft to the distal LCA was then fashioned, and the proximal anomalous LCA was ligated. The patient was restudied at 6 months because of recurrence of symptoms, and recanalization of the anomalous LCA was found. At reoperation, the internal mammary graft was nonfunctional, and suture closure of the orifice of the anomalous LCA was performed. Two years later, the patient died suddenly at

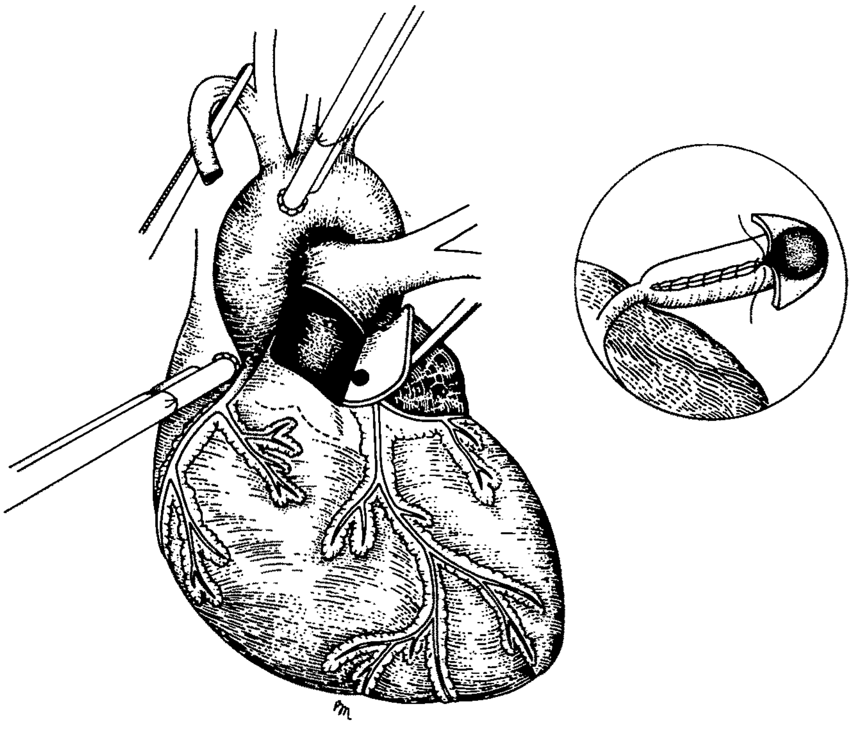

Fig 2. The anomalous left coronary artery is elongated using pulmonary artery wall.

home. Autopsy showed a scarred left ventricle but no other abnormality.

Five patients experienced low cardiac output necessitating inotropic support in the immediate postoperative period. One of these patients required peritoneal dialysis for subsequent renal failure. None of the patients undergoing repair with the new technique had low cardiac output. However, 1 of them showed a transient hemiparesis with complete recovery at late follow-up (Table 2).

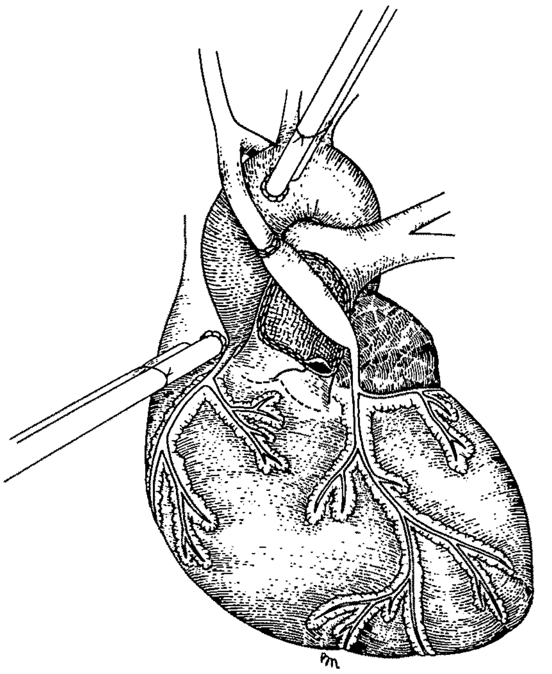

Fig 3. End-to-end anastomosis is accomplished from the right subclavian artery to the pulmonary artery and then to the anomalous left coronary artery.

One patient was lost to follow-up. The remaining 8 survivors were followed for 15 to 61 months postoperatively and were in New York Heart Association functional class I or II. All showed a reduction in cardiac size and the return of a normal ECG. Cardiac function was assessed at late follow-up by cardiac catheterization and angiography, echocardiography, or exercise stress test, and was improved in every patient. Two patent coronary arteries were confirmed in all 6 surviving patients who underwent a procedure designed to establish a two-coronary artery system (Table 3). Three patients demonstrated minor residual mitral regurgitation (see Table 2).

Comment

Various techniques have been proposed for palliation or correction of anomalous LCA. Although no single technique is universally accepted, most surgeons agree that the treatment of choice includes creation of a two-coronary artery system. In our series, six surgical techniques were used over a 15-year period, one of which evolved during that time and represents a new surgical therapy for anomalous LCA.

Postnatal reduction in pulmonary vascular resistance produces early myocardial ischemia secondary to coronary steal in the majority of patients with anomalous LCA. These patients usually are seen with considerable cardiac failure in infancy or early childhood. Therefore, surgical treatment must be designed to safely manage a decompensated myocardium, and the new coronary artery should resist degeneration and have potential for growth.

Previous techniques to surgically repair anomalous

Table 2. Early and Late Postoperative Results

Patient No.	Early Complications		Hospitalization (days)	Follow-up (mo)	Late Complications	NYHA Class
	Cardiac	Noncardiac				
1	Cardiac failure	None	10	66	Angina, recanalization, sudden death	. . .
2	Cardiac failure	None	14	16	Residual MR	II
3	Cardiac failure	None	10	53	None	I
4	Died in OR	. . .	. . .	. . .	. . .	. . .
5	None	None	8	61	None	I
6	Cardiac failure	Renal failure	23	30	Residual MR	I
7	Died in OR	. . .	. . .	. . .	. . .	. . .
8	Cardiac failure	None	10	24	None	I
9	None	None	7	56	Residual MR	I
10	None	Hemiparesis	18	30	None	I
11	None	None	6	15	None	I
12	None	None	9	Lost	. . .	. . .

MR = mitral regurgitation; NYHA = New York Heart Association; OR = operating room.

LCA use cardiopulmonary bypass and require aortic cross-clamping before establishment of the two-coronary artery system. Although cardioplegia provides some protection to the myocardium during these periods, a technique that avoids additional myocardial ischemia before revascularization would be superior. Recent reports describe aortic reimplantation [16] and creation of an aortopulmonary window [17] as surgical treatment for anomalous LCA in infancy and early childhood. Operative death was approximately 20% in these two studies, and in another [18], major postoperative cardiac complications were encountered.

The new technique described here involves pulmonary artery wall extension of the anomalous LCA and anastomosis to the right subclavian artery, does not require aortic cross-clamping, and provides maximal support to the myocardium on cardiopulmonary bypass. Occlusion of the proximal anomalous LCA as soon as bypass is established eliminates coronary steal while the anastomoses are constructed. The procedure requires minimal mobilization of the anomalous LCA and provides pulmonary artery tissue for lengthening the anomalous artery. The pulmonary artery tissue can function as a high-pressure conduit without undergoing degeneration and has the potential for growth. This technique allows for the correction of other intracardiac defects and is also suitable for an anomalous LCA originating from the distal pulmonary artery.

Four patients in the series were surgically treated with this procedure during infancy. Their mean weight was 6.3 kg. As was noted in patients who underwent related repairs, all 4 patients had myocardial ischemia on the ECG, and 3 were seen with cardiac failure. Each of the 4 showed rapid cardiac recovery and had no postoperative cardiac complications. At late follow-up, all were in New York Heart Association functional class I. Two of the

Table 3. Results of Postoperative Cardiac Assessment

Patient No.	ECG Q Wave Depth (mm)		Method of Evaluation	Coronary Artery Anatomy
	Preop	Follow-up		
1	0.2	0.04	Cardiac catheterization	One coronary[a]
2	0.24	0.08	Cardiac catheterization	Two coronaries
3	0.32	0.08	Cardiac catheterization, thallium scan	One coronary
5	0.4	0.04	Exercise test	. . .
6	0.2	0.08	Cardiac catheterization	Two coronaries
8	0.12	0.04	Echocardiography	Two coronaries
9	0.12	0.0	Cardiac catheterization	Two coronaries
10	0.4	0.0	Cardiac catheterization	Two coronaries
11	0.4	0.0	Echocardiography, thallium scan	Two coronaries

[a] This patient died late postoperatively.

patients underwent recatheterization at 30 and 56 months postoperatively, and improved left ventricular function and a patent left coronary artery system were documented.

We conclude that the technique of elongation of the anomalous LCA using pulmonary artery tissue and end-to-end anastomosis to the right subclavian artery on cardiopulmonary bypass without aortic cross-clamping is a viable surgical alternative for repair of an anomalous LCA originating from the pulmonary artery.

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Efficacy and Benefit of Mediastinal Computed Tomography as a Selection Method for Mediastinoscopy

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In 95 consecutive patients with proven or suspected bronchial carcinoma, computed tomographic evaluation of the upper mediastinum for N2 disease was performed prospectively. Patients with positive results underwent mediastinoscopy. Patients with perinodal N2 or N3 disease at mediastinoscopy were not considered candidates for operation. The mediastinum was declared negative only when intraoperative mediastinal lymph node dissection showed tumor-free nodes. Of the 95 patients, 12 had benign lesions, 14 were excluded from further evaluation because the lymph node status of the mediastinum was not proven intraoperatively, and 6 others were excluded from the final evaluation because of violation of the protocol. Twenty-two of the 75 remaining patients had a positive computed tomographic scan and underwent mediastinoscopy. Fourteen patients with positive results were considered to have inoperable disease. Fifty-three patients (70.7%) did not undergo mediastinoscopy. We performed seven probably incomplete resections, two for palliative reasons, and two thoracotomies without resection in patients with N2 disease. A policy of routine mediastinoscopy would have prevented only 5% of the thoracotomies performed in patients with lung cancer.

(Ann Thorac Surg 1989;48:565–7)

In a previous study of patients with lung cancer we [1] compared routine preoperative mediastinoscopy with plain tomography. We found mediastinal metastases (N2) in 64% of the patients by tomography and in 79% by mediastinoscopy. We concluded that 66 of the 85 mediastinoscopies performed were unnecessary or unhelpful. On the premise that N2 metastasis with perinodal growth precludes curative resection, 4 patients were spared an incomplete resection when nodal disease was found by routine mediastinoscopy that had been missed by tomography. Subsequent studies [2–4] indicated that computed tomography may be more sensitive and specific than tomography for the detection of mediastinal metastases. With this background, we undertook another prospective trial to answer the following question: is it justifiable to perform mediastinoscopy electively only in patients who are found to have metastases by computed tomographic (CT) scans?

Material and Methods

All patients admitted to the Department of General and Thoracic Surgery between May 1, 1985, and December 31, 1986, with a proven or suspected bronchial carcinoma that was presumed operable were included in this study. Patients with N3 disease or bulky mediastinal tumors were excluded. Patients who were found to have a nonmalignant histological classification postoperatively were not excluded from the statistical analysis of the efficacy and benefit derived from preoperative diagnostic procedures. All but 1 patient had a preoperative CT scan of the mediastinum.

Computed tomography was performed with the CGR (Companie Général Radiologique) CE 10,000. The slices were 5 mm thick at the hilum and 10 mm elsewhere. Contrast medium was administered intravenously by bolus technique. Evaluation of the CT images was performed by two experienced radiologists. A lymph node was considered to be tumor involved if the diameter exceeded 10 mm.

If the CT scan showed evidence of an N2 or N3 metastasis in the upper mediastinum, a cervical mediastinoscopy was performed. The evaluation included the pretracheal, right and left paratracheal, and tracheobronchial regions and the subcarinal nodes as far as possible. Exploration terminated at the right upper lobe bronchus and the left pulmonary artery.

If the results of mediastinoscopy were negative, thoracotomy was performed. Left anterior mediastinotomy for Botallo's lymph nodes was not done.

Patients in whom perinodal N2 or N3 metastases were found at mediastinoscopy were not considered candidates for operation. Patients who did undergo surgical intervention had a combined lung resection and mediastinal lymph node dissection, which included the lymph nodes at stations 1, 2, 3, 7, and 10 on the right and stations 4, 5, 7, and 10 on the left [5].

The mediastinal findings were defined as positive if lymph node metastases were found by mediastinoscopy

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or thoracotomy. The mediastinal findings were defined as negative if all lymph nodes found at intraoperative lymphadenectomy were negative. Lymphadenectomy was not performed if frozen section of the tumor showed benign disease. Patients who had bronchial carcinoma but did not undergo mediastinal lymphadenectomy were excluded because the study design required lymphadenectomy for the determination of a true negative mediastinal lymph node status.

Results

Of the 95 patients screened for the study, 9 were female and 86 were male. Twenty of the 95 patients were excluded from the final evaluation; 1 did not undergo preoperative mediastinal computed tomography, 14 did not undergo lymphadenectomy for various reasons (age older than 70 years, unfavorable medical condition, unresectability not related to mediastinal metastases), and 5 did not undergo mediastinoscopy in spite of a positive CT scan.

Histological Classifications

In the postoperative pathological evaluation, 12 patients were found to have no malignancy (tuberculomas, inflammatory tumors, lung infarctions). Of the 63 patients with proven bronchial carcinoma and known mediastinal status, 28 (44.4%) had squamous cell carcinoma, 18 (28.6%) had adenocarcinoma, 7 (11.1%) had small cell carcinoma, and 10 (15.9%) had other cell types (large cell, clear cell, and bronchioalveolar carcinomas). Twenty-three had N2 metastasis, which was confirmed in 12 of 31 tumors on the left side and 11 of 32 tumors on the right side.

Computed Tomography

Twenty-two of the fully evaluable group of 75 patients had a positive CT scan. Two of the scans were false-positive, and 20 were confirmed true-positive by mediastinoscopy or lymphadenectomy. There were three false-negative and 50 true-negative scans confirmed by lymphadenectomy or benign disease (sensitivity, 89.3%; specificity, 96.2%).

Mediastinoscopy and Thoracotomy

Twenty-two patients (29.3% of the 75 fully evaluable patients) underwent mediastinoscopy. The indications and results are shown in Table 1. Mediastinoscopy was not performed in 6 patients in spite of a positive CT scan (Table 2).

In 14 of the 75 evaluable patients, the results of mediastinoscopy were positive with perinodal growth, and surgical intervention was not undertaken. We performed thoracotomies on the other 49 patients with bronchial carcinoma and defined mediastinal status, including 9 patients with N2 disease. Lung resection was done in 7 of them, and 2 had exploration and biopsy only. Of these 9 patients, 2 had a false-negative result from mediastinoscopy, and 7 did not undergo mediastinoscopy. Three of those 7 had a false-negative CT scan; the indications for thoracotomy in the other 4 are listed in Table 2.

There was 1 postoperative death 16 days after a sleeve resection of the right upper lobe in a patient with N2 disease.

Table 1. Indications and Results of Mediastinoscopy in 22 Patients

	N2 True-Positive (Mediastinoscopy or Thoracotomy)		
Indication	Mediastinoscopy Positive	Mediastinoscopy False-Negative	N2 True-Negative
CT scan positive (n = 16)	14	2	0
Negative CT scan, positive conventional tomogram	0	0	1
Sleeve resection planned	0	0	2
Oat cell tumor, CT scan negative	0	0	1
Other	0	0	2

CT = computed tomographic.

Comment

Our study evaluating plain tomography versus mediastinoscopy [1] led us to conclude that mediastinoscopy could identify perinodal tumor growth, grossly or microscopically, in more than 80% of patients. Because perinodal growth in the mediastinum has been shown to have a very poor prognosis, thoracotomy for resection in such patients should be avoided [6, 7]. Mediastinoscopy was able to prevent an "unhelpful" thoracotomy in about 80% of the N2 or N3 patients in that series. It is equally clear from the present study and others [6] that mediastinoscopy is very effective for documenting N2 and N3 disease, and saves the patient needless discomfort and disability by preventing thoracotomy without resection. On the other hand, a mediastinoscopy with negative findings, whether true-negative or false-negative, is an invasive procedure that does not contribute directly to treatment and carries a modest risk of complications [1]. If the patient has no malignancy, the procedure is usually

Table 2. Reason for Thoractomy Without Prior Mediastinoscopy in 6 Patients With Positive Computed Tomographic Scan

Reason	Positive Scan	True-Positive at Thoractomy
Palliative pulmonary resection planned	2	1
Isolated left-sided N2 metastasis in aortic arch not reachable with our mediastinoscopy technique	4	3

unnecessary. If its findings are negative in patients with cancer, mediastinoscopy simply confirms that there is no contraindication to thoracotomy.

In terms of health policy planning, the routine use of mediastinoscopy in all patients suspected of having lung cancer adds substantial cost to their management. However, mediastinoscopy reduces the incidence of unnecessary or unhelpful thoracotomy. How then can we best reduce the incidence of unnecessary mediastinoscopy? Preoperative determination of the tissue diagnosis by bronchoscopic or transthoracic needle biopsy may help by focusing the procedure on patients with proven lung cancers or lesions that are highly suspicious on radiographic grounds.

Both plain tomography and computed tomography proved effective in defining mediastinal node status. In our first study [1], the predictive value of a negative plain tomogram of the chest was 73%. A policy of reserving mediastinoscopy for patients with enlarged or indeterminate nodes on tomography would have reduced the incidence of mediastinoscopy by 75%, and might have led to thoracotomy without resection in 16%. (We proved whether it was reasonable to use tomography to select patients for mediastinoscopy.)

In this study, the predictive value of a negative CT scan of the mediastinum was 94%. We performed 22 mediastinoscopies (29.3%). We can logically project that a policy of using the CT scan to select patients for mediastinoscopy would result in 70.7% fewer mediastinoscopies than would a policy of routine mediastinoscopy.

Three of 63 patients with bronchial carcinoma had a false-negative CT scan. If we were to advocate selective mediastinoscopy only for patients with a positive CT scan, 3 (4.8%) would undergo a probably incomplete resection that could otherwise be avoided by a policy of routine cervical mediastinoscopy. These results are superior to those achieved with plain tomography. Computed tomographic scanning provides additional information about the primary tumor, chest wall, pleura, liver, and adrenal glands. On these grounds, it seems preferable to use radiographic staging when it is readily available.

The relatively high number of N2 patients with left-sided tumors and metastasis into station 5 (the Botallo lymph nodes) indicates the advisability of doing either an anterior mediastinotomy or an extended mediastinoscopy when these nodes are enlarged on CT scans. We suggest reformulating the indication for anterior mediastinoscopy or extended mediastinoscopy to include only those patients with enlarged nodes in the subaortic region on the CT scan. Elective mediastinoscopy, using the results of mediastinal computed tomography as the selection criteria, seems a valid way of avoiding unnecessary mediastinoscopies and unhelpful thoracotomies. By using this selection procedure, we were able to avoid about 70% of routine mediastinoscopies, and the risk of unhelpful thoracotomies was kept to about 5%.

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Endobronchial Stenting for Anastomotic Stenosis After Sleeve Resection

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Bronchial anastomotic stenosis is a potentially serious complication of sleeve resection. A Silastic endobronchial stent that prevents progressive narrowing at the anastomotic site is described. Insertion of the stent offers a simple and effective treatment that allows improved pulmonary performance, clearing of infection, and good quality of life. The long-term prognosis, however, remains questionable.

(Ann Thorac Surg 1989;48:568–71)

Survival after sleeve resection for lung tumors in select patients compares favorably with that after pneumonectomy. Morbidity and mortality are reduced while preserving lung tissue and without compromising on tumor clearance. A potentially serious complication of sleeve resection is bronchial anastomotic stenosis, which is due to interrupted bronchial blood supply to the distal bronchial segment and partial anastomotic dehiscence, with granulation tissue formation. There are few therapeutic options available for the treatment of this complication. They include repeated dilations of the stenotic area, immediate resection and reanastomosis, or completion pneumonectomy.

This report describes our clinical experience with 2 patients in whom an endobronchial stent was used to manage bronchial anastomotic stenosis. We discuss the advantages and potential disadvantages of this method.

Material and Methods

Technique

The stent is fashioned using an appropriate length of the vertical limb of a 10- to 14-mm Silastic (Dow Corning) Montgomery T tube, and the rims are made from short segments of a slightly larger Montgomery T tube. The parts are fitted together with silicone glue (Fig 1).

The stent is inserted at bronchoscopy under general anesthesia. Ventilation is maintained by a Venturi jet system. The site and length of the stricture are assessed. With bronchoscopic guidance, the stenotic area is dilated with graduated English bougies. Secretion and pus are evacuated from the distal bronchial tree. The bronchoscopic telescope can then be negotiated through the stricture, and the anatomy beyond can be assessed and biopsy done.

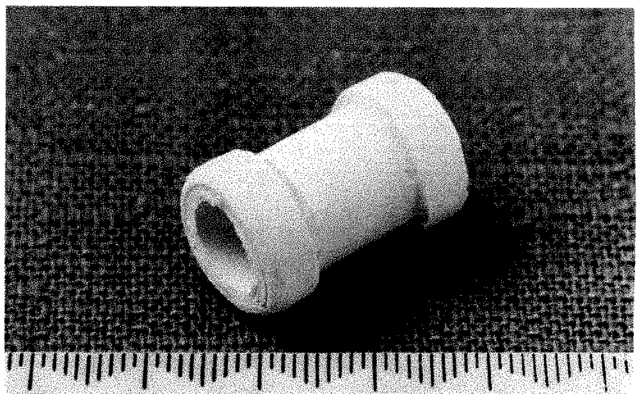

Fig 1. Silastic endobronchial stent.

A fine English bougie (Fig 2) is positioned across the stricture (Fig 3a). The stent of desired size is selected and placed over the bougie (Fig 3b), and is then manipulated through the larynx and into the trachea with a laryngoscope and McGill's forceps. The rigid bronchoscope is used to push the stent to the stricture site (Fig 3c, d). The tip of the bronchoscope is used to keep the stent in a stable position while the fine bougie is withdrawn (Fig 3e). Any fine adjustment can then be done with a grasping forceps through the rigid bronchoscope (Fig 3).

Case Reports

PATIENT 1. A 65-year-old male smoker was given four courses of chemotherapy (cyclophosphamide, vincristine

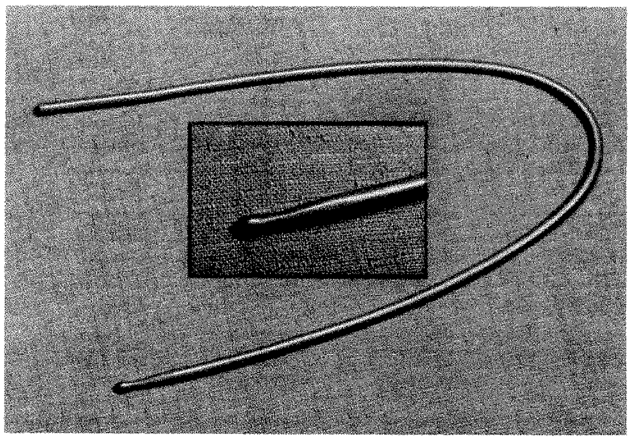

Fig 2. Fine English bougie with a highly malleable stem and a hard plastic tip.

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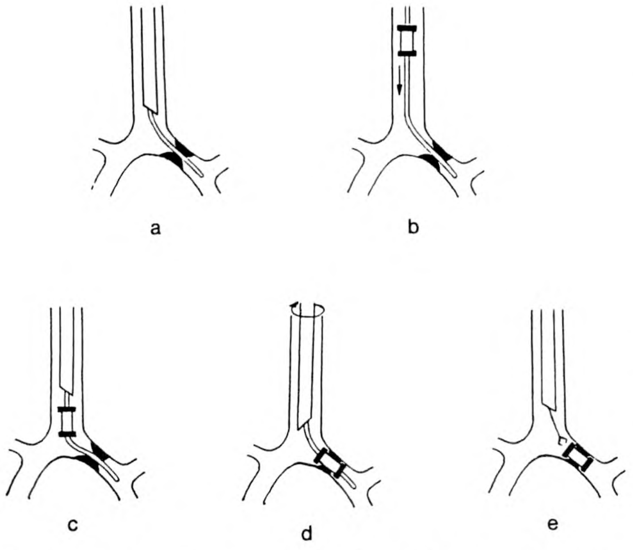

Fig 3. Technique of bronchoscopic endobronchial stenting. See text for details.

sulfate, methotrexate) for small cell carcinoma of the right upper lobe bronchus diagnosed on brush cytology in January 1987. He was well until 8 months later when he was seen with recurrent hemoptysis. Bronchoscopy and repeat biopsy showed squamous carcinoma in the right upper lobe bronchus, and the original cell type was thought to be erroneous.

Computed tomography predicted a normal mediastinum. The lung function was good with a forced expiratory volume in 1 second to forced vital capacity ratio (FEV_1/FVC) of 2.7:4.0. A right upper lobectomy was undertaken. However, the bronchial margin appeared erythematous, and frozen section of this area revealed carcinoma in situ. Sleeve resection of the right main bronchus was carried out, and an end-to-end anastomosis was performed using a continuous suture of 3-0 Prolene. Postoperative evaluation showed resection margins to be clear, and confirmed squamous cell type and N0 status.

Six weeks later, the residual right lung collapsed, and the patient became profoundly hypoxic (Fig 4A). Emergency bronchoscopy demonstrated complete closure at the bronchial anastomotic site with granulation tissue, which was excised with diathermy to establish an adequate lumen. Three weeks later, the patient returned with further collapse of the residual right lung, and a tight fibrous stricture was noted at the site of the sleeve anastomosis ($FEV_1/FVC = 1.6:2.8$). The biopsy specimen showed no malignancy. In an attempt to avoid a completion pneumonectomy, we elected to insert a Silastic endobronchial stent 10 mm in diameter. After this procedure, the chest radiograph showed good aeration of the right lung (Fig 4B), and there was associated symptomatic improvement.

Four weeks later, the stent was changed to a bigger size (12 mm) and was left in position for 6 weeks (Fig 5). At the end of 10 weeks from the time of initial stent placement, the stent was removed, and 4 weeks later, the anastomotic site was assessed. The lumen was considered inadequate, and a 12-mm stent was inserted, which was later replaced by a 14-mm stent. The stent has been well tolerated, and the patient has managed well with good exercise tolerance over the last 15 months ($FEV_1/FVC = 2.7:4.9$).

PATIENT 2. A 55-year-old male nonsmoker with a 4-year history of asthma was seen with hemoptysis. The asthma did not lessen with bronchodilators, and the patient had always maintained it was unilateral. Bronchoscopy revealed a 1-cm polypoid tumor in the left main bronchus (Fig 6). The biopsy specimen demonstrated it to be a squamous papilloma. Computed tomography revealed an endobronchial lesion and no mediastinal abnormality.

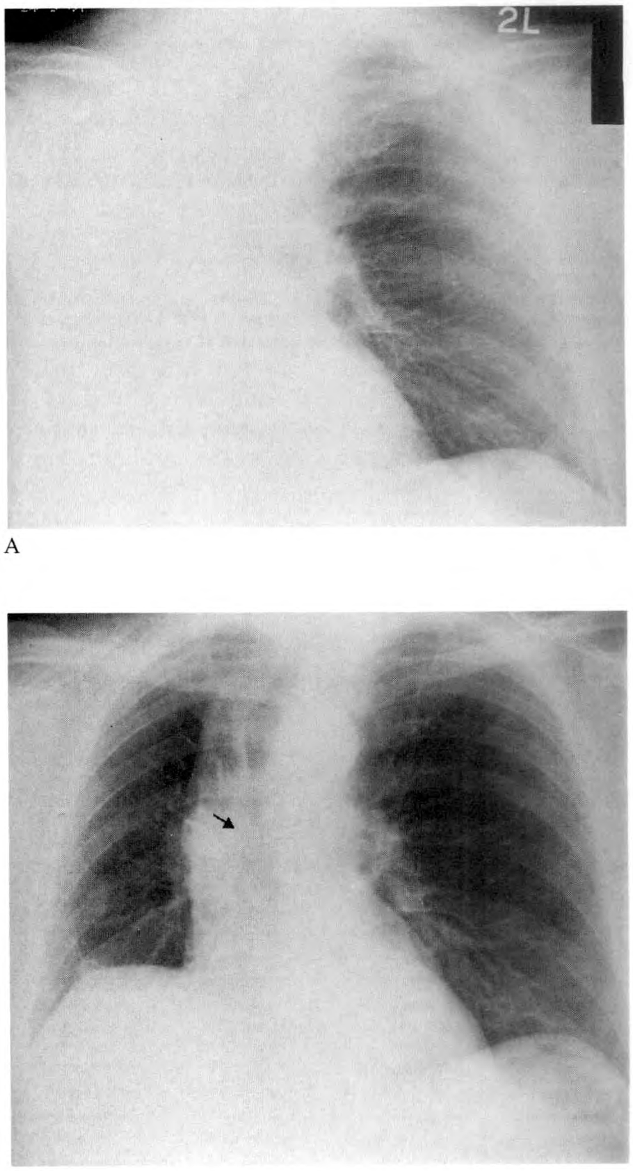

Fig 4. (Patient 1.) Chest roentgenograms of anastomotic stenosis occurring after sleeve resection and treated by endobronchial stenting. (A) Complete collapse of the residual right lung. (B) Stent (arrow) *was placed across the bronchial anastomosis and resulted in reaeration of the remaining lobes on the right.*

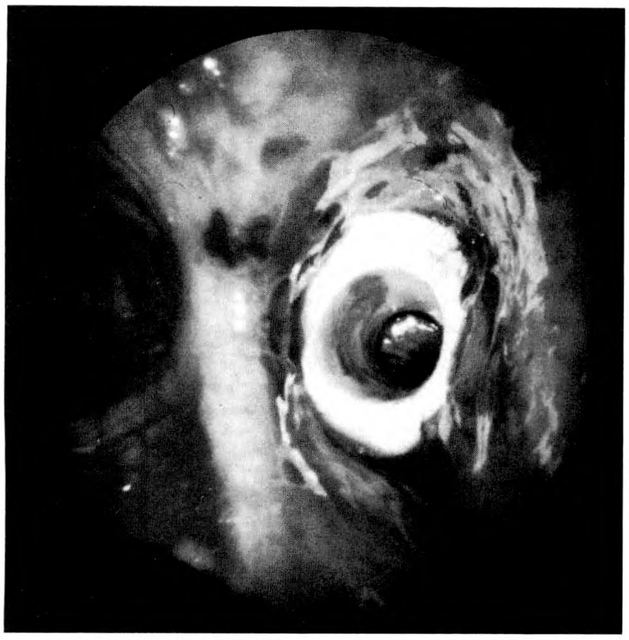

Fig 5. Bronchoscopic view of the bronchial stent in situ.

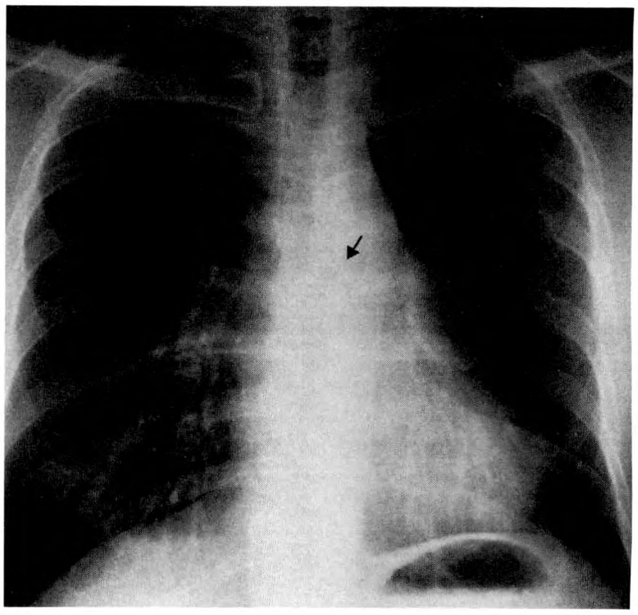

*Fig 7. Bronchial stent (*arrow*), the proximal margin flush with the carina. There was full aeration of the left lung.*

Through a left thoracotomy, sleeve resection of 1.5 cm of the left main bronchus was undertaken with a clear margin. The residual left main bronchus was anastomosed to the carina using a continuous suture of 3-0 Prolene with interrupted 2-0 Prolene sutures at the 6-o'clock and 12-o'clock positions. At the end of the procedure, the anastomosis was checked by bronchoscopy and found to be satisfactory.

Four weeks after sleeve resection, a persistent cough developed, but the patient clinically had normal aeration of the left lung ($FEV_1/FVC = 2.6{:}2.8$). Bronchoscopy revealed edematous mucosa narrowing the anastomosis. The stenotic site was dilated. Two months later, there was absent air entry over the left lung ($FEV_1/FVC = 2.0{:}2.6$). Bronchoscopy disclosed a very tight fibrous stricture at the sleeve resection site, which was opened with a bougie. A biopsy specimen taken at the time was reported to show no tumor. A 12-mm bronchial stent was inserted with the proximal margin flush with the carina (Fig 7). The patient became asymptomatic and has led an active life for the last 12 months ($FEV_1/FVC = 3.1{:}3.4$).

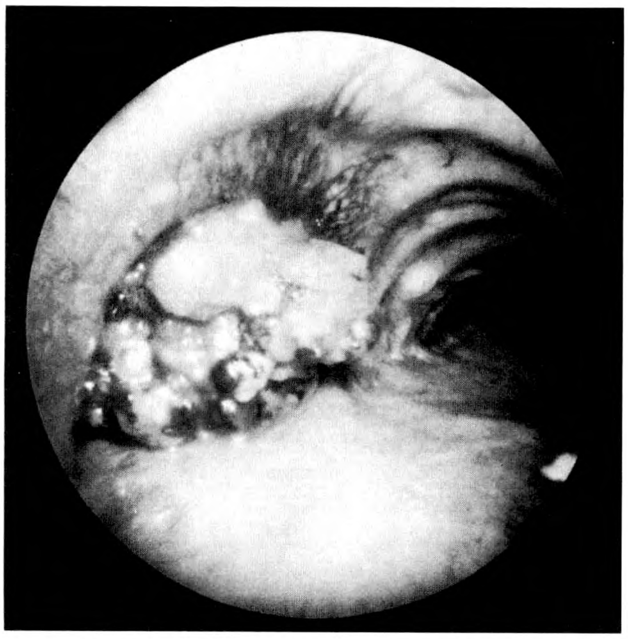

Fig 6. Bronchoscopic view of the polypoid tumor occluding the left main bronchus.

Comment

In select patients, bronchoplastic resection has become an established treatment of bronchogenic carcinoma [1, 2] and benign pulmonary lesions [3] with the advantage of preservation of lung parenchyma. The operative mortality, which ranges from 2% to 7% in most series [1, 2], compares favorably with that of pneumonectomy.

Bronchial healing at the site of the anastomosis is a potential problem. At our institution, the incidence of early and late stricture formation after sleeve resection by one of us (P.G.) is 13% (3/23). We believe such formation is mainly related to interrupted bronchial blood supply to the distal bronchial segment, especially when there is extensive mediastinal dissection. In terms of suture material, it has been suggested that the use of absorbable suture may avoid bronchial irritation, which may be related to protruding nonabsorbable suture material [4]. Continuous Prolene suture was used for the bronchial anastomosis in both of our patients, but no protrusion of suture into the bronchial lumen was found.

Few surgical options are available. Bronchoscopic dilation of the anastomotic stenosis and removal of granulation tissue can reestablish an adequate lumen in some patients. For tight fibrous stricture, repeated dilations at short intervals become unpleasant. Traumatic shearing force as a result of multiple bougienages causes further damage to the bronchial mucosa at the anastomotic site. Early resection and reanastomosis is hazardous because of the marked inflammation around the hilar region. Completion pneumonectomy carries a high risk [5], and the patient may not tolerate the loss of the remaining ipsilateral lung tissue. To avoid repeat dilations and early repeat thoracotomy, it seems appropriate to investigate the use of endobronchial stents for tight anastomotic bronchial strictures.

The purpose of the Silastic endobronchial stent is to prevent progressive narrowing of the lumen, thus allowing maximal expansion and recovery of the residual lung and avoiding early reoperation. The design of the stent is simple, and the stent can be made to whatever size and length required using a silicone Montgomery T tube [6]. Once in place, it is stable and flexible, and has no focal pressure effect. In addition, it can be changed to a bigger size. We have noted no complications resulting from our technique. The Silastic stents have been well tolerated and maintain an adequate bronchial lumen, as demonstrated in our 2 patients. Tube displacement has not occurred.

Despite the apparent lack of ingrowth of respiratory epithelium over the stent surface, distal accumulation of secretion has not presented any problem to our patients. They have better pulmonary performance, coughing, and deep breathing. Frequent changing of the stent has not been necessary.

Use of the endobronchial stent appears to be a desirable stopgap in the treatment of bronchial anastomotic stricture. There is a potential problem with shrinking of tissue once the stent is removed, as shown in our first patient. The alternative, use of an endobronchial stent as a long-term measure, should be considered if the stricture recurs, provided the stent is well tolerated and gives the patient better pulmonary performance and improved quality of life. While the stent maintains the patency of the bronchial lumen, the anastomotic scar tissue undergoes maturation and becomes stiffened. This makes a possible future reoperation less hazardous and reanastomosis feasible. We are also exploring the possibility of long-term stenting using expandable steel stents as an alternative to reoperation [7].

Our experience with the medium-term follow-up of 2 patients encourages us to recommend the use of the Silastic endobronchial stent as a simple and safe treatment for early anastomotic bronchial stricture after sleeve resection.

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Right Ventricular Pressure Dynamics After Operation for Pulmonary Stenosis

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Right ventricular pressure dynamics were monitored during the first 24 hours after operation for relief of pulmonary stenosis in 9 patients with dysplastic pulmonary valves who underwent total valvectomy. Rather than the expected immediate drop in pressure after total removal of the obstruction, right ventricular pressures required 18 to 24 hours to decrease to physiological values. In 7 patients, the pressures decreased steadily, but in 2, there was an increase in pressure immediately postoperatively, followed by a gradual decrease.

(Ann Thorac Surg 1989;48:572–4)

Right ventricular (RV) pressure regression is found at follow-up of patients whose pulmonary stenosis was treated by removal of the fixed obstruction by means of valvotomy or valvectomy [1–7]. However, pressures measured intraoperatively immediately after valvectomy or valvotomy do not show the dramatic regression seen at follow-up [1, 2, 5, 6]. The residual obstruction to flow has been attributed to hypercontraction of the subvalvular hypertrophic RV muscle [2, 8, 9] and to hypertrophy of the crista supraventricularis as well as the parietal and septal infundibular bands [2, 10]. Several authors [10–14] have suggested carrying out muscular resection in addition to the valve procedure, with or without patching the RV outflow tract, when there is infundibular stenosis. Other reports [5–7] advise against routine resection.

The present study was undertaken to isolate the period during which the major RV pressure changes occur. To eliminate any possible influence of residual valvular pulmonary stenosis, all patients studied underwent complete resection of the pulmonary valve and had no fixed residual obstruction that could be found at operation.

Material and Methods

Patient Population

During the period 1975 to 1982, 57 patients underwent an open heart procedure for various types of valvular pulmonary stenosis at the Sheba Medical Center, Tel Hashomer, and the Rambam Medical Center, Haifa. Of these patients, 13 (22.8%) who had dysplastic pulmonary valves and underwent total valvectomy were candidates for this study. Four of these 13 were excluded for technical reasons, thus leaving 9, 4 boys and 5 girls, in the study (Table 1). The mean age at operation was 6½ years.

Methods

Before operation, all patients were studied hemodynamically. Operation was performed using routine open-heart techniques, including moderate hypothermia and cardioplegia. All patients underwent complete excision of the obstructive dysplastic cusps of the pulmonary valve. Inspection and digital examination of the right ventricle showed no annular or aberrant bands obstructing the outflow. In 1 patient (patient 3), the valvular annulus remained narrow after valvectomy and had to be enlarged with a small pericardial patch. Pressures were recorded using a No. 18 polyethylene catheter, which was inserted through the chest wall (transatrially, transarterially, or through the RV wall) into the RV cavity. The catheter was left in place for 24 hours.

Results

There were no deaths in this group of patients. The postoperative course was uneventful except for a sternal infection in 1 patient.

In all patients, RV pressure decreased to physiological values within 18 to 24 hours after operation (Fig 1). One hour postoperatively, mean systolic RV pressure had dropped to 65% of the preoperative mean and continued decreasing steadily. Two patients (patients 1 and 4) showed an increase in systolic RV pressure (9 mm Hg and 33 mm Hg, respectively) at first, followed by the same gradual decrease shown by the other 7 patients. By 24 hours, pressure was stable at a mean systolic pressure of 38.3 mm Hg and a mean systolic RV to left ventricular pressure ratio of 0.35.

Comment

Fixed obstruction to ventricular outflow is the result of malformed cusps, hypoplastic fibrous annuli, or fibrous bands. Dynamic obstruction implies muscular structures, circular or bandlike, within the right ventricle and its outflow tract actively participating in cardiac contraction and causing various degrees of obstruction during the cardiac cycle [3].

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Table 1. Summary of Patient Data Before Operation for Pulmonary Stenosis

Patient No.	Sex	Age	Preoperative Pressures (mm Hg)		RV/LV Pressure Ratio	Previous Surgical Procedures	Associated Malformations	Additional Surgical Procedures
			RV	LV				
1	F	4 yr	150	110	1.36	Closed valvotomy at age 2 wk	Patent foramen ovale	Closure of patent foramen ovale
2	M	5 yr	125	110	1.13	Closed valvotomy at age 4 mo	. . .	. . .
3	F	6 yr	120	122	0.98	Open valvotomy	Noonan's syndrome	Transannular patch
4	F	5 yr	110	110	1.00	. . .	. . .	. . .
5	M	7 mo	120	120	1.00	. . .	. . .	. . .
6	M	2 yr	200	100	2.00	Closed valvotomy at age 8 days	. . .	. . .
7	F	29 yr	148	110	1.34	. . .	Noonan's syndrome	. . .
8	M	6 yr	92	90	1.02	. . .	Noonan's syndrome, patent foramen ovale	Closure of patent foramen ovale
9	F	1 h	105	98	1.07	. . .	. . .	. . .

LV = left ventricle; RV = right ventricle.

The persistence of high RV pressure during the immediate postoperative period seems to be related to two factors: (1) an outpouring of endogenous catecholamines [15, 16], which raises the pressure in the right ventricle more than in the left ventricle, very likely in proportion to the large hyperdynamic muscle mass of the right ventricle, and (2) early contraction of an obstructing zone, namely, the infundibulum, before the right ventricle empties [3, 4, 12, 13, 17]. Bearing in mind that regression of the obstructing RV muscle requires 3 to 20 months, as seen in electrocardiographic studies [2, 5], it seems reasonable that within the first 18 to 24 hours, the muscle begins to undergo inactivation with progressively decreasing contraction. The pressure decrease is the first necessary step toward regression of the excess muscle mass. This biological process can be viewed as the reverse process of hypertrophy [8, 9].

Growing understanding of RV outflow tract hemodynamics leads us to concur with those authors [2–7] who recommend that we not be tempted to alter a simple valvotomy or valvectomy for relief of pulmonary stenosis by resecting RV muscle should there be a high systolic pressure immediately after bypass. Follow-up cardiac catheterization shows nearly normal to normal RV pressure in almost all of these patients. Both Engle and associates [2] and Brock [3] reported pressure reduction over time without infundibulectomy. Awariefe and colleagues [6] found a reduction in RV pressure of approximately 50% intraoperatively after relief of the obstruction, and noted a further decrease at follow-up catheterization 1 to 2 years later.

In a review of 78 pediatric patients treated surgically for pulmonary stenosis, Griffith and co-workers [5] found that intraoperative (ie, after valvectomy or valvotomy) RV pressures decreased, but the decrease occurred at different rates depending on anatomical differences in the ratios of the infundibular diameter in systole to the valvular ring diameter. At later follow-up, pressures were at physiological levels. The early differences in pressure decreases were not predictive. Arendrup and associates

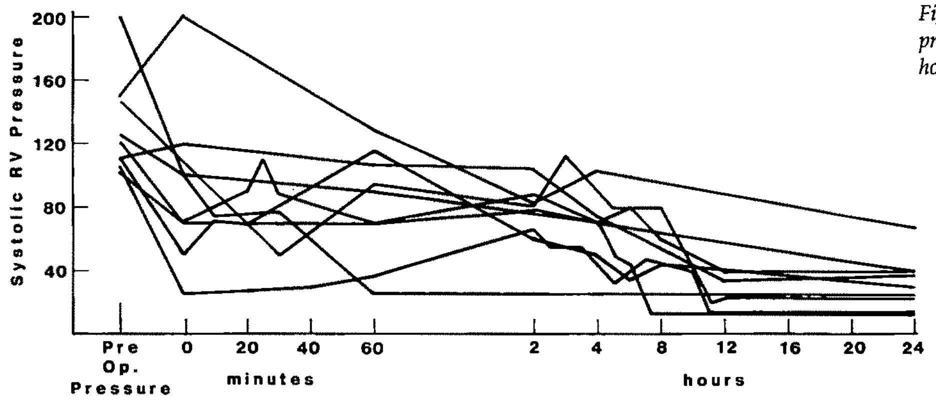

Fig 1. Systolic right ventricular (RV) *pressure preoperatively and up to 24 hours postoperatively.*

[7] reported that in 14 of their patients with infundibular as well as valvular pulmonary stenosis, 1 year after valvotomy, mean RV pressure was 30 mm Hg; the results with valvotomy alone were satisfactory in patients with simple secondary infundibular stenosis.

We have studied RV pressure changes within the 24 hours immediately after operation. In a preliminary report based on 12 patients who underwent either valvotomy or valvectomy, we [18] found that RV pressures had reached physiological levels within that time. Kittle and colleagues [13] had less clear-cut results in 6 patients with pulmonary stenosis studied up to 105 hours. The present study reveals that RV pressure requires up to the first 18 to 24 hours postoperatively to regress. Therefore, provided no residual fixed obstruction exists, surgical procedures to reduce RV outflow tract muscle in patients such as ours are not justified. There is a striking resemblance in the findings of the present study in terms of the pattern of RV pressure regression to those in patients with tetralogy of Fallot, in whom conservative infundibulectomy was shown to be as successful as more radical muscle resection [19].

In summary, this study of RV pressure in patients who have undergone total pulmonary valve resection for relief of pulmonary stenosis shows a regression of pressure to physiological values within the first 24 hours postoperatively. The model of pulmonary valvectomy enabled us to study dynamic RV obstruction alone after removal of the fixed area of narrowing. Based on the results of this study, we conclude that additional surgical procedures to the RV outflow tract muscle are not justified in patients in whom no residual fixed obstruction exists.

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Hypoxemia After Intraluminal Oxygen Line Obstruction During Cardiopulmonary Bypass

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A patient sustained an episode of hypoxemia during cardiopulmonary bypass. Investigation of the extracorporeal circuit after successful resolution of the problem showed that a white, crystalline substance later identified as mannitol occluded the oxygen supply line to a bubble oxygenator. The management and subsequent investigation of the problem are presented.

(Ann Thorac Surg 1989;48:575–6)

The development and clinical utilization of extracorporeal cardiopulmonary bypass techniques constitute a major advance in modern medicine. In 50 years, procedures have advanced from the experimental laboratory to early clinical trials fraught with hazard and complexity to procedures that are common, generally safe, and effective. Clinical techniques have improved, as has the design of circuits and devices. The biomaterials in contact with the blood of the patient have also improved. At present, clinical use of blood oxygenators is generally safe. Although many different blood oxygenators are currently available, design defects do not appear to constitute a serious threat to patient safety. The incidence of harmful effects, errors, failures, or problems attributable to blood oxygenators is difficult to ascertain, but life-threatening complications are rare and episodic in nature. Considering the fundamental complexity of an extracorporeal circuit, the frequency of life-threatening complications is surprisingly low [1].

We present a case report, management, and subsequent investigation of a patient who developed a life-threatening complication during cardiopulmonary bypass; hypoxemia occurred because of obstruction of gas flow to a bubble oxygenator.

A 54-year-old man weighing 86 kg with triple-vessel coronary artery disease and left ventricular aneurysm underwent aortocoronary bypass grafting, left ventricular aneurysm resection, and insertion of intraaortic balloon pump. A Bentley BEN-10 B bubble oxygenator was used in the extracorporeal circuit. After an uneventful bypass, the patient was weaned at 70 minutes elapsed time. One gram of calcium chloride was administered to facilitate separation. Bypass had to be reinstituted to permit repair of a distal graft anastomosis. At 50 minutes elapsed time of the second bypass, the venous oxygen saturation suddenly decreased from 70% to 58%. The arterial blood was dark. Oxygen flow was increased from 2.5 to 6 L/min without improvement in color or saturation. The sound of gas being released through the pressure relief valve of the oxygen source was clearly audible. A decision was made to terminate bypass, and mechanical ventilation of the lungs with oxygen was started. Epinephrine (100 μg) was administered, and an infusion of dopamine (10 μg/kg/min) was started. The patient was separated from bypass, and adequate cardiac output was maintained with dopamine (10 μg/kg) and intraaortic balloon counterpulsation.

The oxygenator was examined after the procedure. A white crystalloid substance was obstructing the oxygen inlet port of the oxygenator. The length of the occlusion was 1 cm. Scattered deposits of the same material were visible on the walls of the oxygenator gas chamber and on the undersurface of the gas sparger plate. A sample of the material was subjected to infrared analysis and mass spectroscopy. A sagittal section of the oxygenator was obtained. Infrared analysis determined that the occlusive material was mannitol. This was confirmed by mass spectroscopy. Sagittal section of the oxygenator revealed that all gas and fluid chambers were intact and all fluid paths were patent.

These conditions were simulated in an unused discarded cardiopulmonary bypass circuit with a Bentley BEN-10 B bubble oxygenator. The circuit was primed with crystalloid solution. Mannitol was then added from the cardiotomy reservoir. The oxygen supply was disconnected at the source during the addition of mannitol. Mannitol flowed into the oxygen tubing. The line was reconnected, and oxygen flow was started. At five minutes, a white precipitate began to form (Fig 1). At 25 minutes, the precipitate was almost occlusive (Figs 2, 3).

Comment

Hypoxemia during cardiopulmonary bypass may be a life-threatening event. Light anesthesia and inadequate muscle relaxation cause mild hypoxemia because of increased peripheral oxygen consumption [2]. Leaks in the oxygen supply line are a potential cause of hypoxemia, which may be mild or severe [3]. Oxygenator failure is a rare event, but design and manufacturing defects have been reported [1, 4]. Gas line obstruction is a potential cause of hypoxemia. Hypoxemia secondary to intraluminal obstruction of the oxygen supply has not been reported.

Mannitol is an osmotic diuretic commonly used in

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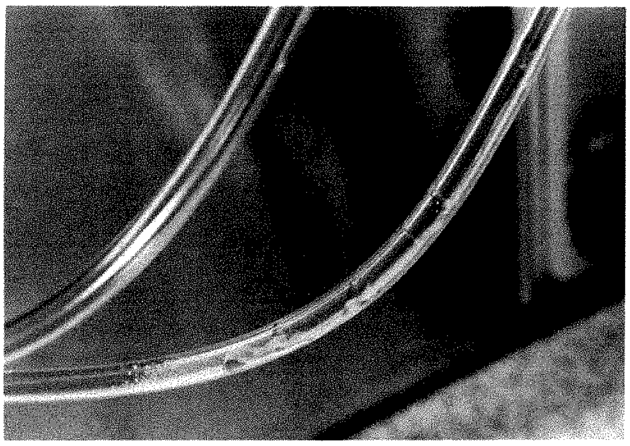

Fig 1. Flocculant precipitate formed five minutes after oxygen flow in tubing containing mannitol was started.

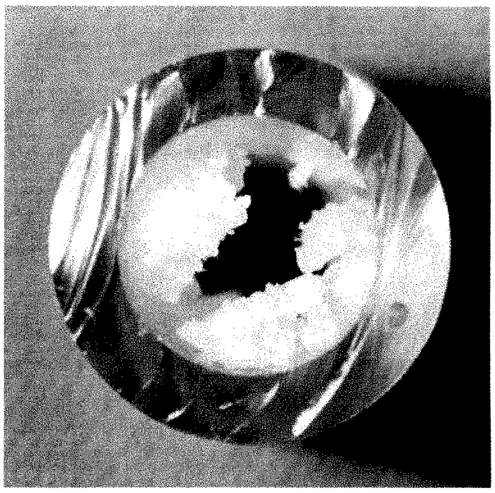

Fig 3. Cross-section of oxygen tubing at site of maximum precipitate formation.

association with cardiopulmonary bypass to enhance urine flow. Solutions of mannitol are chemically stable, but in concentrations of 15% or greater, mannitol may crystallize when exposed to low temperature or polyvinyl chloride surfaces. A heavy, white flocculant precipitate forms, acting as a nucleus for rapid rate crystallization. In our patient, 20% mannitol flowed into the oxygen tubing. This occurred at a time when the oxygen tubing was not connected to the oxygen source. The oxygen tubing was looped in such a way that the nadir of the tubing was lower than the cardiotomy return point of the oxygenator. The oxygen tubing was then properly connected, and gas flow was started through the tubing. Several hours after mannitol was added to the prime, crystallization occurred and occluded the oxygen tubing.

One may conclude from this near mishap that the routine preoperative check list of a cardiopulmonary bypass circuit should include a thorough examination of the oxygen supply line for leaks, disconnections, and the presence of substances in the tubing. Mannitol should be diluted before administration into the prime or should be administered into the circuit after cardiopulmonary bypass is started.

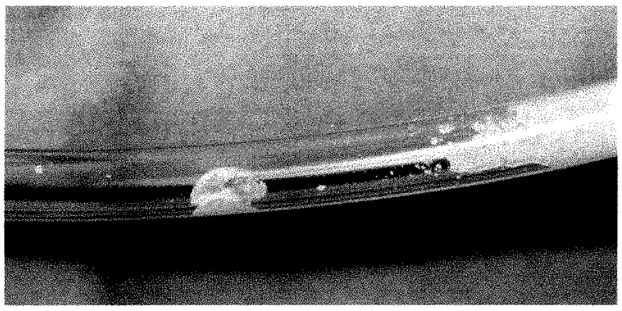

Fig 2. Near occlusion of oxygen tubing 25 minutes after oxygen flow was started.

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Late Failure of Double-Inlet Left Ventricle Septation: Treatment by Orthotopic Heart Transplantation

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We report a patient in whom orthotopic heart transplantation was performed after late failure of ventricular septation for double-inlet left ventricle. This case shows that orthotopic heart transplantation represents a valid therapeutic alternative in children with previous correction of complex congenital heart defects not amenable to further intracardiac repair.

(Ann Thorac Surg 1989;48:577–8)

Definitive repair by ventricular septation in patients with double-inlet ventricle has proved successful in selected cases [1]; however, data on the long-term fate of this operation are still limited [2]. We report a case of late failure of ventricular septation treated by orthotopic heart transplantation.

A 16-year-old boy was referred to our department in January 1987 because of signs of biventricular failure after ventricular septation performed elsewhere 5 years earlier for repair of double-inlet left ventricle with discordant atrioventricular and ventriculoarterial connections (single left ventricle, type A3) with left-sided and anterior aorta. The postoperative period had been complicated by complete atrioventricular block requiring permanent pacemaker implantation and by congestive ventricular failure treated with prolonged inotropic support. He had been discharged 25 days after operation on chronic digoxin and diuretic agents.

During the next 5 years, the patient showed retarded growth, restricted physical activity, and borderline cardiac performance requiring frequent hospitalization. After an episode of severe cardiac failure, complicated by liver dysfunction and ascites, he was admitted to our hospital for hemodynamic evaluation. Cardiac catheterization revealed hypertension in the right ventricular chamber (80/16 mm Hg) with a transpulmonary gradient of 58 mm Hg and increased end-diastolic pressures in both right and left ventricular chambers (16 and 18 mm Hg, respectively). Angiography showed massive right atrioventricular valve regurgitation and a severe subpulmonary stenosis created by a rightward bulging of the interventricular patch, which restricted the right ventricular chamber to an almost virtual cavity (Fig 1). Because of the presence of biventricular dysfunction, his condition was not considered suitable for a modified Fontan procedure. He was therefore scheduled for heart transplantation as the only remaining surgical option.

Orthotopic heart transplantation was performed 5 months later using the heart of a 9-year-old compatible donor; total ischemic time was 164 minutes, and aortic cross-clamp time was 24 minutes. The anomalous position of the great arteries did not alter the standard surgical technique markedly, requiring only a more extensive mobilization of the great vessels to bring the right-sided and posterior pulmonary artery to its final left-sided and anterior position, typical of the usual arterial arrangement in the presence of a normal conotruncus. This maneuver was easily performed by taking advantage of our routine harvesting technique, which includes removal of the entire length of the main pulmonary artery with its bifurcation. The patient was weaned off cardiopulmonary bypass without any difficulty, and the early postoperative period was uneventful.

According to our protocol of treatment for heart transplantation in pediatric patients, immunosuppression consisted of cyclosporin A, beginning at a whole blood level of about 1,000 ng/L using the radioimmunoassay method (Sandoz kit, Sandoz, Basel, Switzerland), progressively tapered to 400 ng/L over the first 3 postoperative months, and azathioprine to maintain a white blood cell count of about 5,000/μL. Chronic oral steroids were not used. Four weeks after operation, the patient experienced an episode of moderate acute rejection, which regressed after a three-day course of intravenous methylprednisolone. At present, 16 months after heart transplantation, he is asymptomatic and enjoys a life without limitations; current echocardiographic and hemodynamic studies show excellent graft performance.

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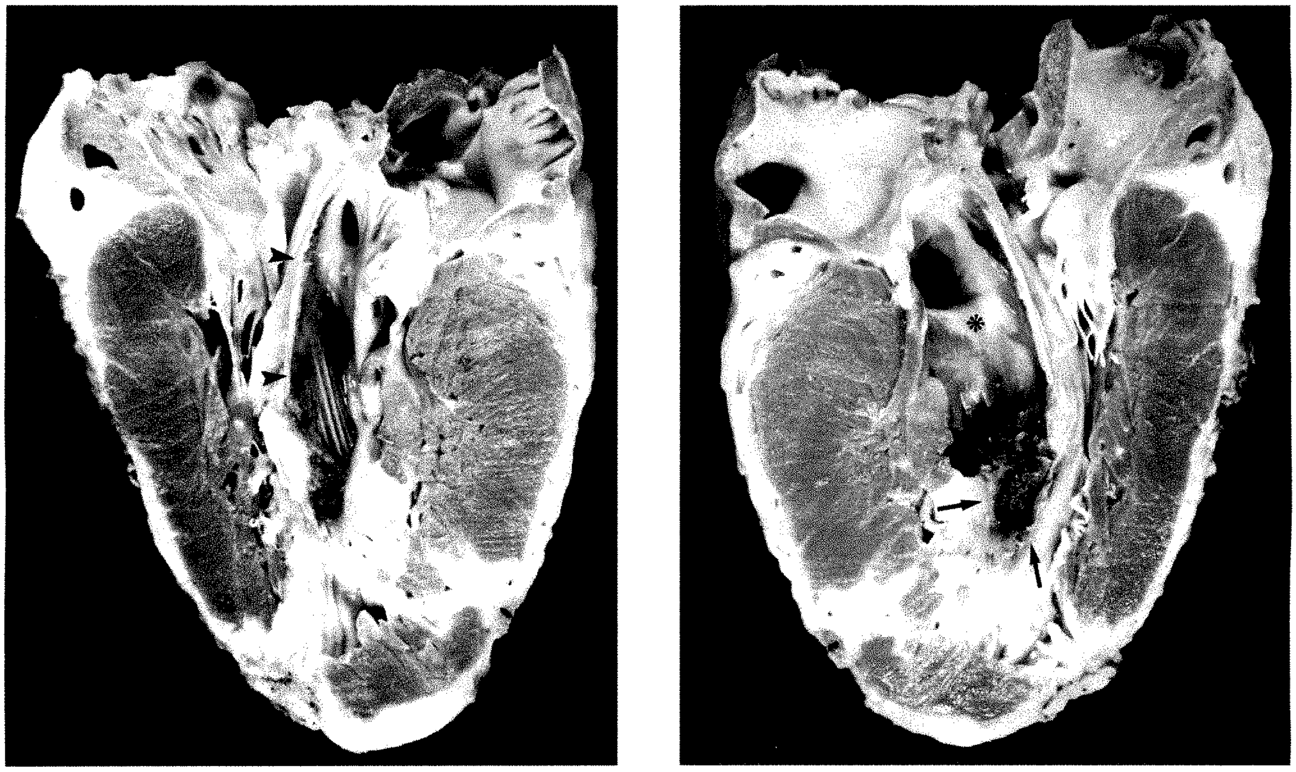

A B

Fig 1. Heart specimen cut in four-chamber view. (A) Posterior view shows reconstruction of the atrioventricular connection of the double-inlet left ventricle with a large Dacron patch. Right-sided bulging of the intraventricular patch (arrowheads) *obstructed the right ventricular chamber. (B) Anterior view shows subpulmonary obstruction owing to bulging of the intraventricular patch, thrombosis at the apex of the left ventricular chamber* (arrows) *in correspondence with the ventriculotomy scar, and a widely patent outlet foramen (*). (Reprinted from Thiene G, Calabrese F, Faggian G, et al. Native heart pathology in cardiac transplantation. In: Gallucci V, Bortolotti U, Faggian G, Mazzucco A, eds. Heart and heart-lung transplantation update. Firenze: USES Edizioni Scientifiche, 1988:133–54, with permission.)*

Comment

Although ventricular septation has been successfully performed in patients with double-inlet ventricle, the operative mortality remains high and the late results uncertain [2]. Pacifico and colleagues [2], in reviewing the University of Alabama experience with 36 consecutive ventricular septation operations, reported an actuarial survival at 3 years of 57% and that major causes of surgical failure were ventricular patch dehiscence, atrioventricular valve incompetence, and pacemaker malfunction.

Failure of ventricular septation in our patient was caused by an extreme rightward bulging of the large interventricular patch, creating a severe, fixed subpulmonary stenosis. Such unfavorable evolution of this operation has apparently never been reported but may become more evident as follow-up of these patients progresses. When we first examined the patient, any further intracardiac repair, such as a modified Fontan procedure, was contraindicated by the presence of a major degree of "left ventricular" dysfunction, leaving orthotopic heart transplantation as the ultimate surgical choice.

Orthotopic heart transplantation was recently reported in a patient with univentricular heart and suggested as alternative treatment in select patients with double-inlet–type atrioventricular connection [3]. The excellent surgical result and the dramatic improvement in quality of life observed in our patient confirm that orthotopic heart transplantation can also be consistently considered for patients previously treated by intracardiac repair for complex congenital heart malformations [4].

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Intraoperative Use of Dual-Chamber Demand Pacemakers for Open Heart Operations

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The availability of external atrioventricular sequential pacemakers has improved the management of patients with sinus bradycardia, junctional rhythm, and atrioventricular block. However, these pacemakers are of less value in patients with postoperative heart block and accelerated atrial rhythms. The temporary use of a modified explanted dual-chamber demand pacemaker may counteract that problem by providing atrially triggered, P-wave–synchronous ventricular pacing. We report 2 patients in whom the temporary use of the dual-chamber demand pacemaker greatly facilitated weaning from cardiopulmonary bypass after coronary artery bypass grafting.

(Ann Thorac Surg 1989;48:579–81)

The use of external pacemakers after cardiac operations to provide atrial or atrioventricular (AV) sequential pacing is helpful in the management of complete heart block, bradycardias, and atrial, junctional, and ventricular ectopic beats [1, 2]. Permanent implantable dual-chamber demand (DDD) pacemakers are widely used, and external DDD pacemakers have been modified for temporary use [3, 4]. We report the use of a modified explanted DDD pacemaker to provide temporary external pacing in 2 patients.

Modification of Pacemaker

A Pacesetter 283 explanted DDD unipolar pacemaker was placed in a small utility box, and external binding posts were attached for atrial and ventricular electrodes. Ventricular and atrial output were set at 5 V with a pulse width of 0.6 ms, ventricular sensitivity was set at 3.0 mV, atrial sensing was set at 0.5 mV, rate limits were set at 90 and 129 beats/min, and AV interval was set at 140 ms. These settings could be changed with an external programmer, which remained at the bedside of the patient while the pacemaker was in use.

Case Reports

Patient 1

A 43-year-old woman underwent five-vessel coronary artery bypass grafting. While she was still on cardiopulmonary bypass third-degree AV block developed, and AV sequential pacing was begun using bipolar atrial and ventricular leads. Weaning from cardiopulmonary bypass was unsuccessful, and administration of epinephrine (4 μg/min) was started. Her atrial rhythm accelerated, and third-degree AV block continued. The high rates required to achieve both atrial and ventricular capture prevented the use of AV sequential pacing. Cardiac output and blood pressure were low; pulmonary artery pressures were elevated. The modified DDD pacemaker was placed externally, and weaning was successful with P-wave–synchronized ventricular pacing at 124 beats/min; pulmonary artery and systemic blood pressures became satisfactory. Normal sinus rhythm returned on the second postoperative day, with first-degree AV block and left anterior fascicular block. The modified DDD pacemaker was removed.

Patient 2

A 50-year-old man underwent four-vessel coronary artery bypass grafting. When weaning from cardiopulmonary bypass was begun, his normal sinus rhythm converted to third-degree AV block, with accelerated atrial rhythm and multifocal premature ventricular contractions. Ventricular tachycardia responded to cardioversion, and cardiopulmonary bypass was reinstituted. Epinephrine (6 μg/min) was infused, and an intraaortic balloon assist device was placed. The modified DDD pacemaker was placed externally, and P-wave–synchronous ventricular pacing at 128 beats/min was satisfactory. The patient's hemodynamic status stabilized, and weaning was successful. On the second postoperative day, normal sinus rhythm returned, with a left anterior fascicular block, and the pacemaker was removed.

Comment

The physiological advantages of temporary AV sequential pacemakers in open heart surgical patients are clear [2, 5]. In patients with normal AV conduction, properly timed atrial systole augments cardiac output by improving ventricular stroke volume and end-diastolic pressure [6] and is 30% more effective than ventricular pacing alone [7]. With compromised left ventricular compliance secondary to myocardial infarction or cardiopulmonary bypass, optimal AV delay in AV sequential pacing may also improve cardiac output, even when AV conduction is maintained

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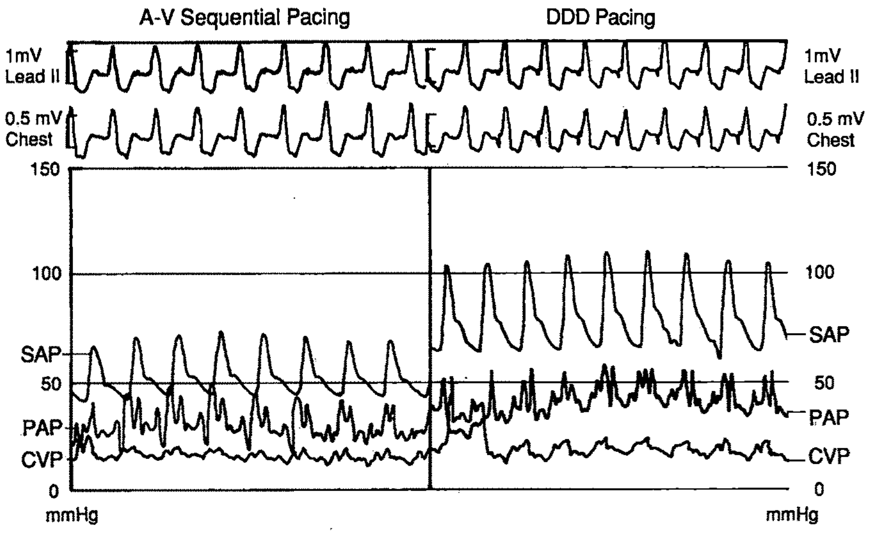

Fig 1. (Left) Simultaneous electrocardiogram with systemic arterial pressure (SAP), pulmonary arterial pressure (PAP), and central venous pressure (CVP) during ineffective atrioventricular (A-V) sequential pacing. (Right) The same tracings four minutes later with P-wave–synchronous ventricular pacing, showing substantial improvement in systemic arterial pressure.

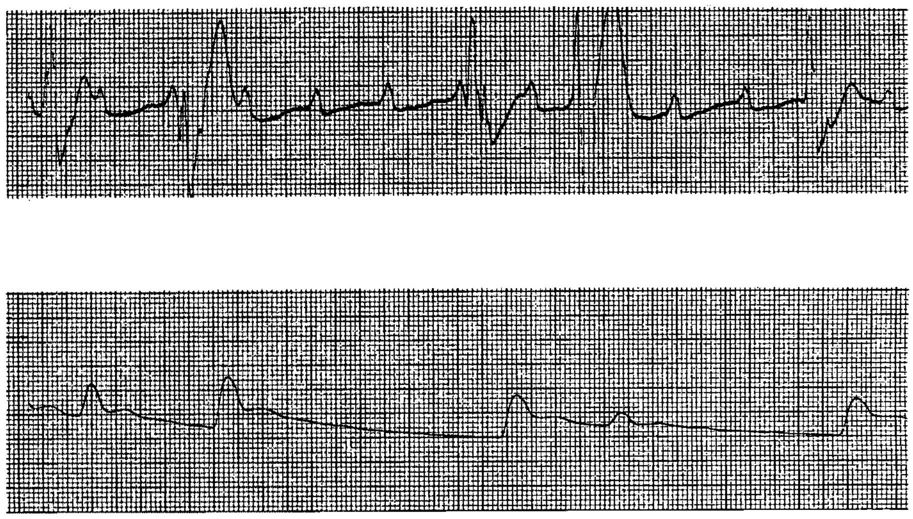

Fig 2. Simultaneous electrocardiogram (top) and systemic arterial waveform (bottom) showing oversensing with loss of ventricular pacing and decreased systemic arterial pressure with external dual-chamber demand pacemaker.

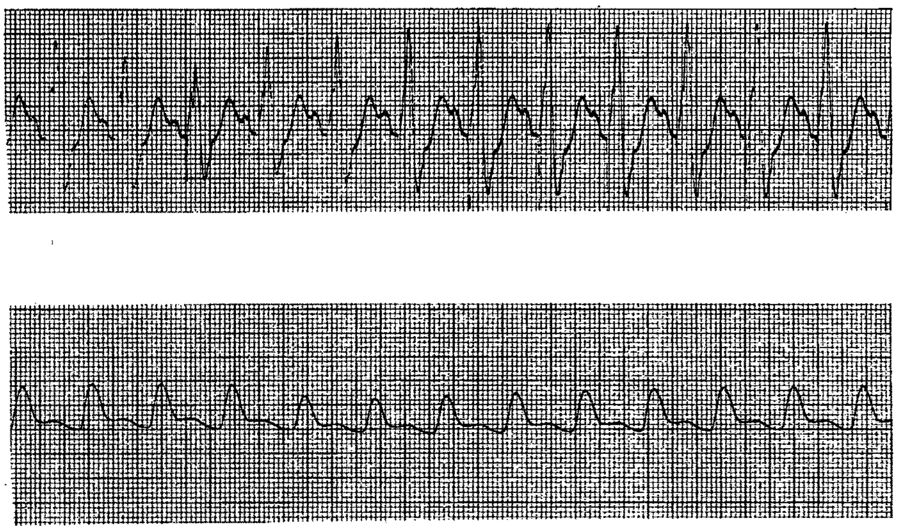

Fig 3. Simultaneous electrocardiogram (top) and systemic arterial waveform (bottom) showing appropriate P-wave–synchronous ventricular pacing with external dual-chamber demand pacemaker.

[7]. Accelerated atrial rhythm in the presence of third-degree AV block precludes optimal cardiac output with simple ventricular or AV sequential pacing; in these patients, P-wave–synchronous ventricular pacing can be beneficial [3, 4] (Fig 1). Atrial pacing is also helpful when a long AV delay causes suboptimal augmentation of stroke volume and "atrially induced mitral valve regurgitation" [6]. Finally, AV sequential pacing may also suppress atrial and junctional tachycardias, ventricular ectopy, and reentrant mechanisms [1]. However, when tachycardias exceed those controllable with AV sequential pacing, suppression of junctional and ventricular dysrhythmias is more practical with P-wave–synchronous ventricular pacing.

With the currently available external sequential pacemakers (eg, Medtronic 5330), "cross-talk" may enable the atrial pulse to suppress the ventricular channel if ventricular sensitivity is low (oversensing). Because the atrial channel is unable to sense atrial systole, pacer-induced dysrhythmias also may result. Temporary use of a modified, explanted DDD pacemaker has proved helpful in such situations [3, 4]. Although the problems of atrial and ventricular oversensing may make the DDD pacemaker difficult to use, proper programming of output and sensing variables may improve the results (Figs 2, 3). Further investigation into this problem is warranted so that external DDD pacemakers will become available, if only for temporary use. Improved pacemaker design should reduce the incidence of oversensing of atrial and ventricular channels and will ensure safe temporary use of these pacemakers.

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Esophageal Rupture Complicated by a Gastrocardiac Fistula: Review of the Literature and Report of a Successfully Treated Case

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A 62-year-old man developed a fistula between the right ventricle and the stomach after Thal fundic patching of an emetogenic rupture of the esophagus. He underwent emergency surgical correction of the fistula and survived.

(Ann Thorac Surg 1989;48:582–3)

Fistulous communications between the heart and the upper gastrointestinal tract are extremely rare and in the past have been uniformly fatal. We report a successfully treated case and a brief review of the literature.

A 62-year-old man had an 18-hour history of vomiting and abdominal pain. His past medical history included severe peptic ulcer disease and alcoholism. Contrast films revealed a ruptured esophagus just above the gastroesophageal junction. At thoracotomy, a 7-cm tear of the esophagus was repaired with a gastric patch (Thal procedure) and drained with bilateral chest tubes.

Three weeks after the original operation, massive upper gastrointestinal bleeding developed. Selective total visceral angiography, including venous phase, failed to disclose the site of the hemorrhage. At laparotomy, the source of bleeding was found to be a fistula between the fundus of the stomach and the right ventricle. Through an emergency left anterior thoracotomy, adhesions between the heart and stomach were broken with blunt dissection. The hole in the right ventricle was located in the lower portion of the chamber and was about 3 mm in diameter (Fig 1); it was repaired with pledgetted sutures. An end-cervical esophagostomy, distal esophagogastrectomy, and feeding jejunostomy were performed. Forty-two units of blood were required. Antibiotics were continued for 30 days and total parenteral nutrition was continued for 6 weeks. Gastrointestinal continuity was restored with a substernal colon transplant and jejunal pouch (Fig 2). Further recovery was uneventful and, when last interviewed, the patient was without dietary restrictions and had gained approximately 20 pounds. He is alive and well 3 years later.

Comment

Although a fistula between the upper gastrointestinal tract and the heart had been reported as early as 1880, only 15 well-documented cases of this entity have been described in the world literature to date. All previous cases have been diagnosed at autopsy.

In 1886, Finney reported a case of a 19-year-old man whose gastric ulcer had perforated through the diaphragm and into the left ventricle of the heart [1]. He was seen with exsanguinating hemorrhage and died. Including Finney's report, there have been nine bona fide reports of fistulas between the upper gastrointestinal tract and the left ventricle [1–3]. All were reported to be secondary to peptic ulcer disease. In only 1 patient had a

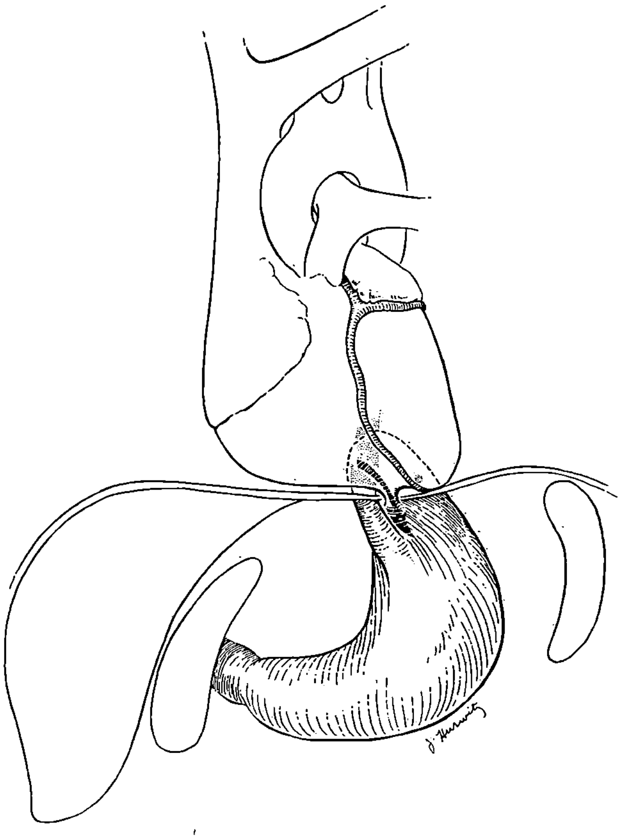

Fig 1. Gastrocardiac fistula; blood flows from the right ventricle to the stomach (arrow).

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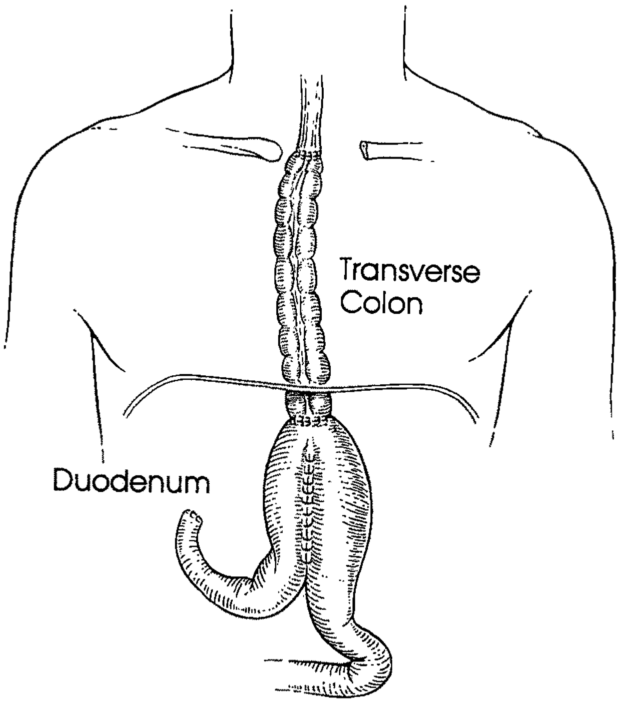

Fig 2. Gastrointestinal continuity was restored with a substernal colon transplant and jejunal pouch.

gastrectomy been performed [3]. Six cases of gastrointestinal–left atrial fistula have been reported [1, 4]. Three of these have involved anastomotic ulcers; the rest were esophageal. Two of the patients with esophageal ulcers had reflux and stricture. The other patient with esophageal ulcer had had trauma to the gastrointestinal cardiac fistula, as well as some preexisting esophagogastric disease.

Some Clinical Clues

Initially, all these patients had upper gastrointestinal bleeds. In addition, several bizarre symptoms may also be present. Ladnyuk and colleagues [3] reported a patient with gastric–left ventricular fistula who experienced bizarre substernal buzzing sensations before having exsanguinating hemorrhage. All 6 reported patients with fistulas to the left atrium had some type of neurological symptoms, most of them focal in nature [1, 4], and all who were subjected to autopsy had either multiple microabscesses of the brain or food emboli in the cerebral circulation.

Thus, the diagnosis of gastrointestinal–cardiac fistula should be considered in a patient with a history of upper gastrointestinal disease or operation who has upper gastrointestinal bleeding and focal neurological deficits. Negative angiography results with concomitant massive gastrointestinal hemorrhage should also suggest this lesion.

This is, to our knowledge, the first report of a fistula between the right ventricle and the gastrointestinal tract. Right ventricular lesions may be more survivable than left ventricular lesions for two reasons. Any food emboli entering the right ventricle would usually become lodged in the pulmonary capillaries rather than the systemic vessels; in fact, our patient had no focal neurological deficits. In addition, the right ventricular pressure is much lower than that of the left ventricle and, although the hemorrhage was quite dramatic, it might have been much worse if the fistula had been to the left ventricle.

The advantages of a buttressed repair in the treatment of the perforated esophagus have been described by many investigators [5–7]. The various techniques employed use stomach, pleura, pericardial fat, diaphragm, and intercostal muscle. Each method has its advocates, and no one technique has been proved superior.

Use of the Thal fundic patch in such cases has received general approval, although at least one similar, catastrophic complication has been reported. In describing a case of gastric aortic fistula subsequent to a Thal procedure, Harwood and co-workers [8] noted that the anatomical situation created by this procedure is not unlike an incarcerated paraesophageal hernia. Such hernias have a reported 30% incidence of ulceration [8]. We therefore do not recommend use of a Thal patch in such cases unless alternative buttressing tissues are not available.

Summary

Fifteen well-documented cases of gastrointestinal–cardiac fistula have been reported previously. All patients died. We report, to our knowledge, the first case of a patient who survived such a fistula. He required aggressive surgical treatment and prolonged critical care. Patients may have a typical symptom complex depending on the site of the fistula.

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The Danger in Skin Grafting the Bare Mediastinum After Sternectomy for Postcoronary Bypass Dehiscence

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We describe a patient who underwent coronary bypass grafting, after which severe mediastinitis and sternal osteomyelitis occurred. Repair after sternectomy was undertaken with a rectus-abdominis myocutaneous flap. The distal fifth of the flap underwent necrosis and was replaced by a meshed split-thickness skin graft. A year later, a clip marking one of the bypass grafts nearly eroded through the skin graft, endangering the bypass graft. The skin graft was removed by abrasion, and the bypass graft was covered with a pectoralis muscle flap. We recommend that skin grafting of a granulating wound over coronary artery bypass grafts be avoided if possible.

(Ann Thorac Surg 1989;48:584–6)

Repair of sternal wound dehiscence with infection after midsternotomy incisions has been well described in the thoracic and plastic surgery literature over the last 10 years [1–4]. The advocated method of repair includes thorough bone and cartilage debridement and coverage with muscle flaps, either the pectoralis or the rectus abdominis muscle [5–7]. Other methods are applied if these muscles are not available or if the defect is too small to use a muscle.

In cases in which skin shortage is apparent, the flap is either taken as a myocutaneous flap or preferably as a muscle flap covered with a skin graft. We have used these methods in 70 patients over the past 5 years [8]. In 3 of them, part or all of the bare mediastinum had to be covered with a skin graft: in 1 very sick patient, the graft was used as a salvage procedure, and in 2 others grafts were used to cover areas exposed due to partial necrosis of rectus abdominis myocutaneous flaps and dehiscence of the wound. One of these is described here.

A 55-year-old man had quadruple coronary artery bypass grafting using three veins and the left internal mammary artery. Early sternal dehiscence occurred, and the attempt to reclose the sternum failed. Severe mediastinitis developed and the patient was in grave condition. After prolonged antibiotic treatment and hyperalimentation, a one-stage repair was performed, which included debridement of all the sternum and some of the ribs and coverage with a right rectus abdominis myocutaneous flap. A hematoma developed postoperatively beneath the distal end of the flap, which then underwent partial necrosis. The necrotic part was excised, and the granulation tissue growing in its place was covered with a split-thickness meshed skin graft (Fig 1). The patient recovered satisfactorily and was discharged.

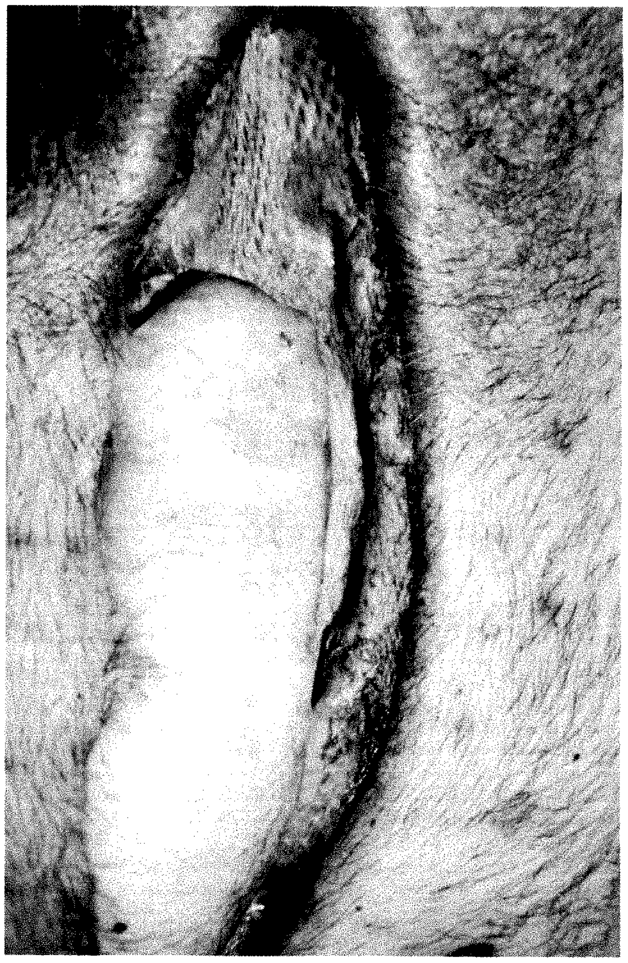

Fig 1. Anterior chest wall with a myocutaneous flap in its center. Skin grafted area is apparent at its proximal end (May 1986).

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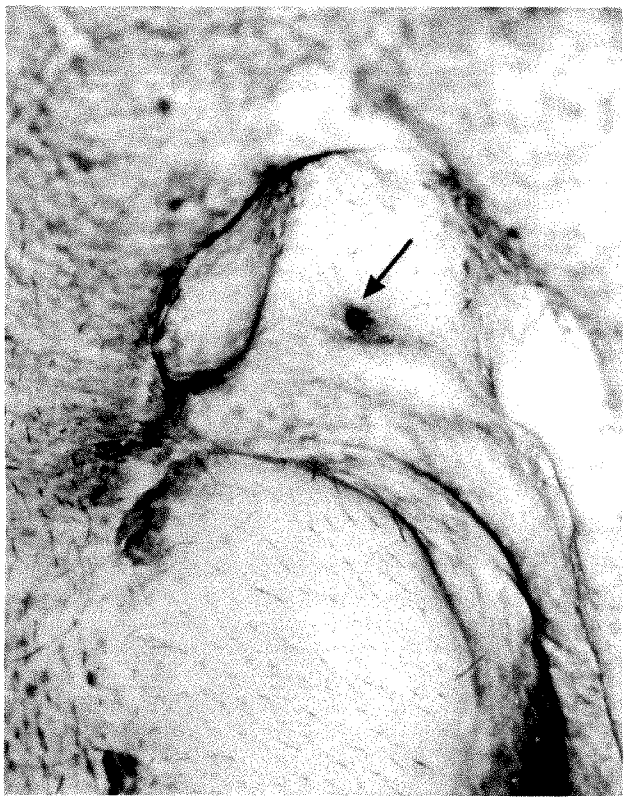

Fig 2. *Bluish bulge in middle of the grafted area (June 1987).*

At one of the patient's routine follow-up visits, a tiny bluish bulge was discovered in the center of the skin grafted area (Fig 2). It was first interpreted to be a small hematoma or a foreign body reaction. Fortunately, the insertion of a forceps to explore the bulge was avoided when it was suggested that it might be the wall of a bypass graft eroding through the skin graft. A simple chest radiogram revealed a silver clip beneath the thinned-out skin graft. An emergency angiogram showed the proximity between the ring marking the vein graft, the graft itself, and the skin surface (Fig 3). The vein graft clearly was covered with a very thin layer of skin only and was endangered by the slightest trauma unless covered with a thick viable layer of tissue. The patient underwent operation in which the entire skin-grafted area was dermabraded with a diamond mechanical dermabrader except for the bulged area, which was left untouched. A pectoralis muscle was raised as a turnover flap to cover the nearly exposed bypass graft. The muscle was covered with a skin graft. The 12-month postsurgical period was uneventful (Fig 4).

Comment

The use of skin grafts to cover the granulating surface of the mediastinum after removal of the sternal bone was reported before the era of myocutaneous flaps [9], and has even been reported in the recent literature [10].

We have used skin grafts on rare occasions, either as a salvage procedure or to cover areas of partial muscle flap necrosis. The patient we report had a complication—the impending erosion of a bypass graft through the skin graft—which made us reluctant to use another skin graft on granulation tissue covering a bypass graft. In such cases, we now make every effort to find different solutions, such as the omentum or other available muscles, even as a second-stage procedure if the patient is too sick to undergo a major operation at the first session. If the granulating defect is small, it should be left to contract and close by secondary intention.

In a literature search, we found no description of any such complication. Neither did we find descriptions of other complications after similar operations. However, we have heard of a case of traumatic rupture of the aorta after blunt trauma in a patient who had a skin graft replacing the entire sternum and part of his rib cage. In one of our

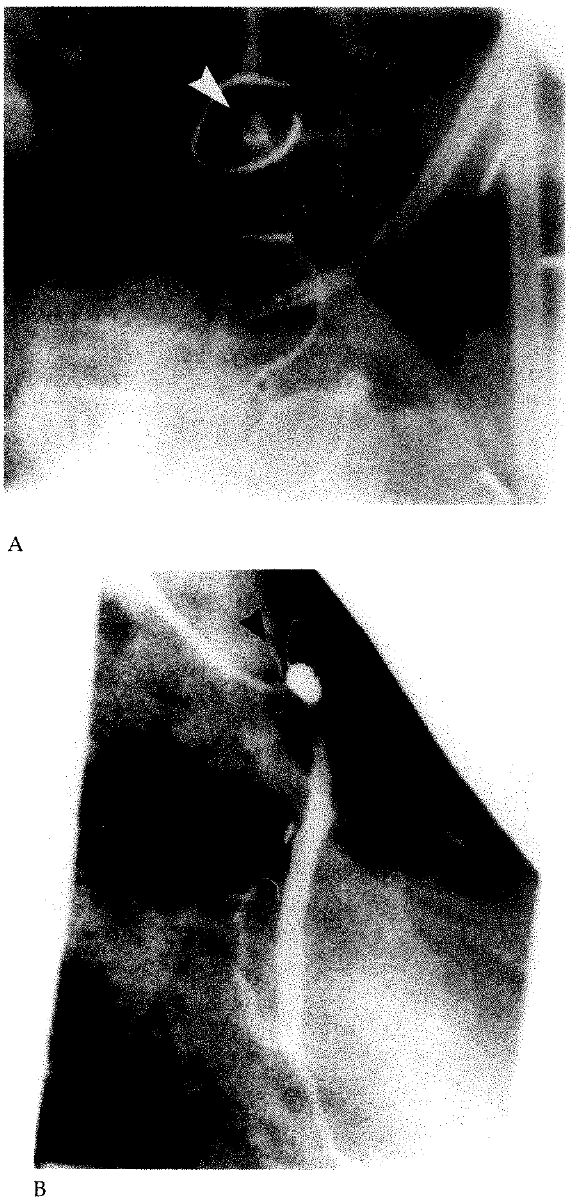

Fig 3. *Angiogram. (A) Anteroposterior view: The open metal thread* (arrow) *was placed on the skin surface around the protruding part of the cut marking ring. (B) Right anterior oblique view during injection to obtuse marginal branch of circumflex coronary artery; metal ring* (arrow) *is close to surface.*

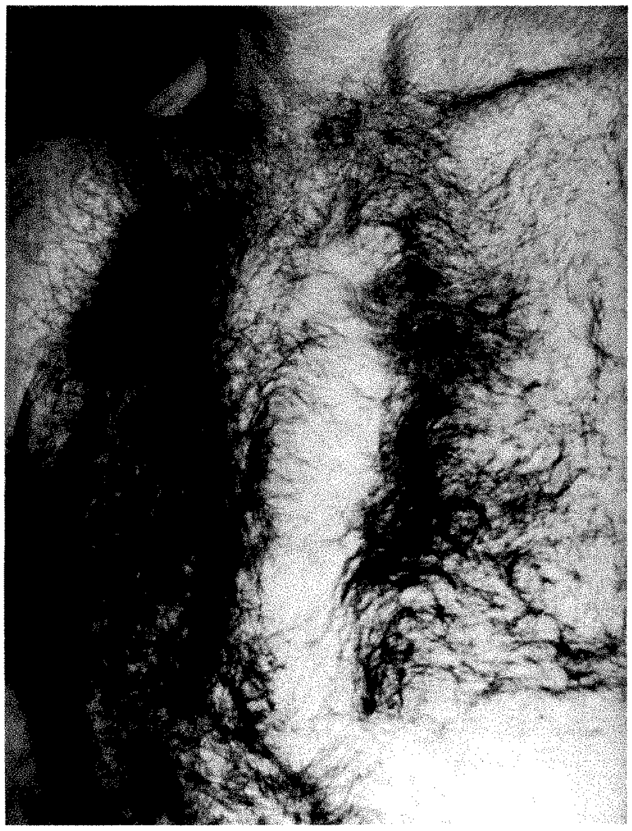

Fig 4. One year after dermabrasion of skin graft: coverage with pectoralis turnover flap and a skin graft (July 1988).

patients with a similar chest wall defect, an acrylic shield was fitted to prevent any blunt trauma to the unprotected mediastinum.

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Truncus Arteriosus With Interrupted Aortic Arch: Successful Correction in a Neonate

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Truncus arteriosus (type II) with interrupted aortic arch (type B) was successfully repaired at 11 days of age using anterior translocation of the pulmonary arteries, resection of the ductus arteriosus, and direct anastomosis between the descending aorta and truncus. This technique permitted wide reconstruction of the aortic arch and minimized the number of suture lines. It also positioned the right ventricle–pulmonary artery conduit anteriorly, which may simplify its subsequent replacement.

(Ann Thorac Surg 1989;48:587–9)

Truncus arteriosus with interrupted aortic arch has been corrected in young children [1–3], and a few infants have survived repair after 3 months of age [4–7]. However, because most babies with this combination of cardiac malformations die in the first weeks of life [8], earlier operation is necessary. We describe successful primary correction of this condition in a patient during the neonatal period.

A male infant weighing 3.4 kg was mildly cyanotic after uncomplicated delivery at 38 weeks of gestation and subsequently required oxygen therapy for respiratory distress. When the infant was five hours old, a murmur was heard; cardiac assessment on the following day showed weak pulses in the left arm and both legs. Real-time echocardiography demonstrated truncus arteriosus with separate origins of adjacent pulmonary arteries (type II) and interruption of the aortic arch distal to the left carotid artery (type B). Treatment with intravenous prostaglandin E_1 stabilized the infant's condition, and cardiac catheterization with angiography on the third day of life confirmed the echocardiographic diagnosis (Fig 1). The infant was observed for several days while mild jaundice resolved; during this time, a trial of withholding prostaglandin established that the ductus would not remain patent.

Complete repair was undertaken through a midline sternal incision when the patient was aged 11 days. Profound hypothermia (15°C) was induced on cardiopulmonary bypass using a single arterial cannula positioned low on the ascending aorta and venous drainage from a single cannula in the right atrium. The branch pulmonary arteries were snared during cooling, and the heart was decompressed through a small right ventriculotomy. The circulation was arrested for a single period of approximately 66 minutes, during which time the brachiocephalic vessels were occluded and the arterial cannula was removed.

The patent ductus arteriosus was first divided just beyond the origin of the pulmonary arteries. The pulmonary arteries were then excised from the posterior aspect of the truncus and dissected to the lung hila, as in the "French maneuver" for anatomical correction of transposed great vessels. All ductal tissue was resected, and the extensively mobilized descending aorta and left subclavian artery were anastomosed posteriorly to the defect in the truncus (Fig 2). This suture line lay close to the left coronary artery ostium. Closure of the ventricular septal defect and placement of a 12-mm composite Dacron–aortic homograft conduit between the right ventricle and pulmonary arteries completed the repair (Fig 3).

Inotropic support (dobutamine, 5 μg/kg/min, and dopamine, 2.5 μg/kg/min) was necessary for five days postoperatively, during which time the infant had good hemodynamics and excellent foot pulses. Pulmonary function, however, was severely compromised, and he required 23 days of ventilation with pleural drainage for multiple pneumothoraces, bilateral serous effusions, and a left chylothorax. These complications all responded to conservative treatment; the patient began to feed well and gain weight. He was discharged from the hospital at age 2 months. Postoperative echocardiography showed normal left ventricular size and function, a widely patent aortic anastomosis, and a good position of the valved extracardiac conduit.

Comment

The simplest method of arch repair in truncus arteriosus with interrupted aortic arch is to retain the patent arterial duct, which already connects the ascending and descending aorta through the truncus arteriosus [1, 3]. In this patient, however, ductal patency was dependent on prostaglandin and therefore unreliable in the long term. The distance between the ascending and descending aorta, as used by Fujiwara and colleagues [6, 7] for arch reconstruc-

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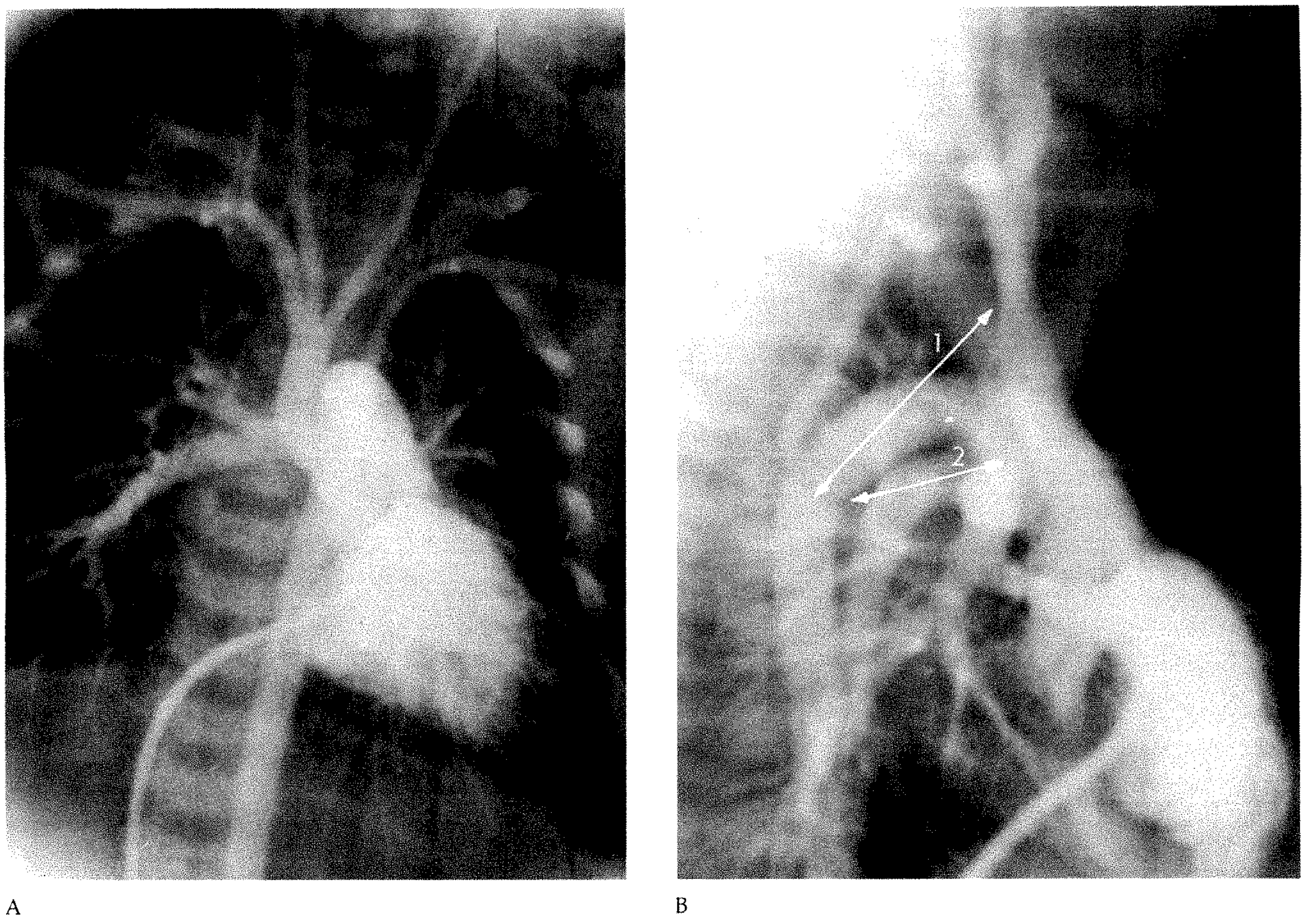

Fig 1. Anteroposterior (A) and lateral (B) views of the left ventricular angiogram showing truncus arteriosus with the descending aorta arising from a large patent ductus arteriosus. The truncus also gives off the pulmonary arteries and ascending aorta, which divides into innominate and left common carotid branches. For arch reconstruction by direct anastomosis, the distance from the descending aorta to the posterior truncus (2) is less than the distance to the ascending aorta (1).

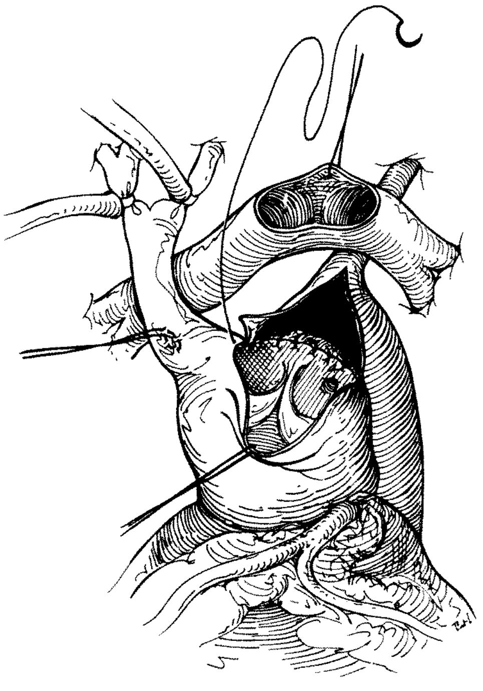

Fig 2. Operative procedure. The pulmonary arteries were detached from the truncus and retracted superiorly, providing access for a posterior anastomosis between the truncus and descending aorta.

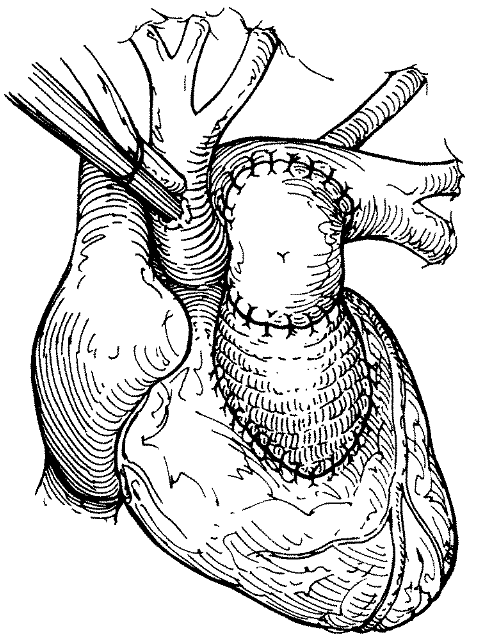

Fig 3. Completed repair. The 12-mm homograft conduit lies in front of the aortic arch reconstruction.

tion by direct anastomosis, appeared to be about equal to the distance from the truncus to the descending aorta (Fig 1). Thus, we found direct anastomosis to the truncus also was possible after the pulmonary arteries had been mobilized. This technique avoided any limitation of systemic blood flow by the small ascending aorta and also incorporated the defect that resulted from excision of the pulmonary arteries, thereby obviating a second incision in the distal aorta and separate closure of the posterior truncus. Although compression of the left main bronchus and distortion of the truncal valve were recognized as potential hazards, these appear to have been prevented, at least in the short term, by adequate mobilization of the vessels. A posterior anastomosis also ensured ample space for the right ventricle to pulmonary artery conduit in its usual position to the left of the aorta, thus permitting use of the incision made to detach the pulmonary arteries from the truncus for the distal anastomosis.

Optimal timing of surgical intervention for truncus arteriosus with interrupted aortic arch is uncertain. Although an older patient may tolerate cardiopulmonary bypass better, the likelihood of pulmonary vascular disease, infection, and malnourishment also increases with the passage of time. Having established that the ductus would not remain patent and having previously lost similar patients owing to rapid decompensation in uncontrollable heart failure or attempted palliative operations, we elected to perform complete repair as soon as the patient's neonatal problems had resolved.

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Giant Benign Mesothelioma

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Pleural mesothelioma is a rare neoplasm that is usually highly malignant, but benign mesotheliomas do occur; approximately 400 cases are described in the literature. We report the case of a young woman with a massive benign mesothelioma that filled the entire left hemithorax but was successfully resected with full reexpansion of the lung.

(Ann Thorac Surg 1989;48:590–1)

Mesotheliomas are rare tumors arising from the mesothelial lining cells of the pleura, peritoneum, and pericardium. Pleural mesothelioma is generally malignant and is associated with asbestos exposure. Approximately 400 cases of benign pleural mesotheliomas have been reported in the world literature. We report the case of a patient with a benign mesothelioma notable for its massive size.

The patient is a 20-year-old Polynesian woman from the island of Maduro in the Marshall Islands. She had noted exertional dyspnea and vague substernal and left anterior chest pain beginning about 1 year before she was admitted to the hospital. A chest roentgenogram showed that the entire left lung field was opacified. Four months before her admission, a purified protein derivative skin test for tuberculosis was positive at 18 mm. The patient was empirically started on chemotherapy with isoniazid, rifampin, and ethambutol. Lack of clinical improvement led to her referral to our hospital.

At the time of admission to our hospital, she continued to complain of left-sided chest pain and dyspnea on exertion. In addition, she began having cough productive of yellow sputum. Physical examination was remarkable for absent breath sounds on the left with dullness to percussion. She had no clubbing or long bone tenderness. Serum chemistry levels were normal; her glucose level was 106 mg/dL. The hemoglobin and hematocrit were within normal limits, and the white blood cell count was 5,100/μL. A chest roentgenogram confirmed the presence of a mass filling the entire left hemithorax (Fig 1). Computed tomography of the chest showed an inhomogeneous density in the left chest with marked compression of the trachea, heart, and other mediastinal structures (Fig 2). Air bronchograms showed the left lung to be collapsed. Arterial blood gas while the patient was breathing room air was within normal limits, but spirometry showed a severe restrictive pulmonary defect with a vital capacity of only 990 cm^3. Bronchoscopy showed slight rightward deviation of the trachea and complete obstruction from extrinsic compression of the left main bronchus at the level of the left upper lobe bronchus. A transthoracic TruCut needle biopsy of the mass showed benign fibrous tissue.

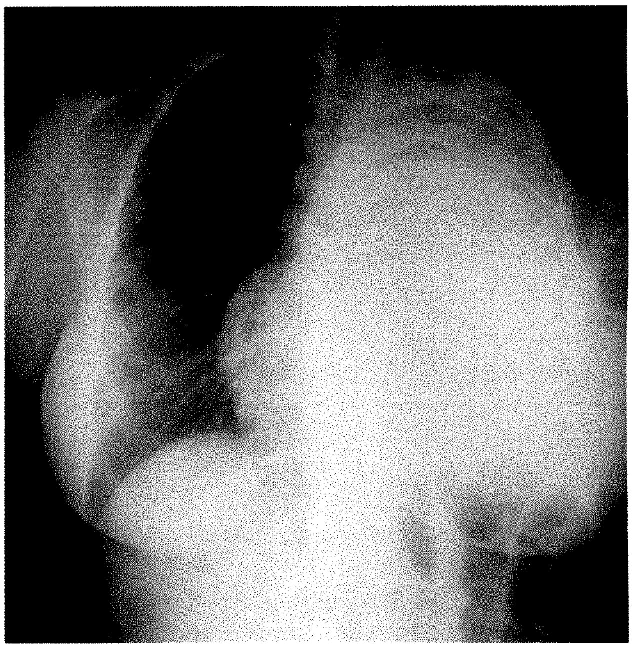

Fig 1. Preoperative chest roentgenogram shows mass involving entire left hemithorax with mediastinal shift.

Resection of the tumor was accomplished through a left posterolateral thoracotomy through the bed of the fifth rib. The tumor was arising from the posterior parietal pleura on a wide-based 5-cm pedicle extending from the second to the fifth ribs. The tumor was excised completely together with a 6 × 6-cm segment of posterior ribs two through five. The pedicle, which had a rich vascular supply, was ligated with both electrocautery and suture ligatures. The mass measured 21 × 16 × 9 cm and weighed 1,460 g. The left lung completely reexpanded after the tumor was removed from the left hemithorax. A chest roentgenogram on the tenth postoperative day showed relief of the mediastinal compression and reexpansion of the lung (Fig 3). The patient tolerated the procedure well and was discharged on the fourteenth

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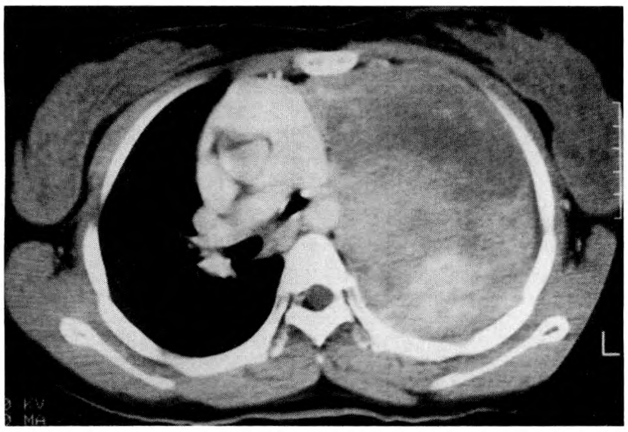

Fig 2. Computed tomographic scan of the chest shows huge left thoracic mass compressing the heart past the midline.

postoperative day. Pathological diagnosis of the tumor was benign localized fibrous mesothelioma.

Comment

Although mesothelioma is a rare disease (about 1,500 new cases a year), its incidence is increasing. Unlike malignant mesothelioma, benign mesothelioma appears to be unrelated to asbestos exposure [1]. Mesothelioma exists in an epithelial (diffuse) form and a fibrosarcomatous (localized) form [2]. The epithelial form is uniformly malignant and rapidly fatal. The localized form is usually considered benign, but malignant cases of localized mesothelioma have been reported [3].

Benign mesothelioma is often first detected as an asymptomatic mass on a chest roentgenogram obtained for another purpose. Rarely the mass fills the entire hemithorax, as in our case. Other common presentations include cough, chest pain, and dyspnea. Digital clubbing is noted in about one third of patients, particularly with tumors greater than 10 cm. Only 1 case of malignant localized mesothelioma was associated with clubbing [4].

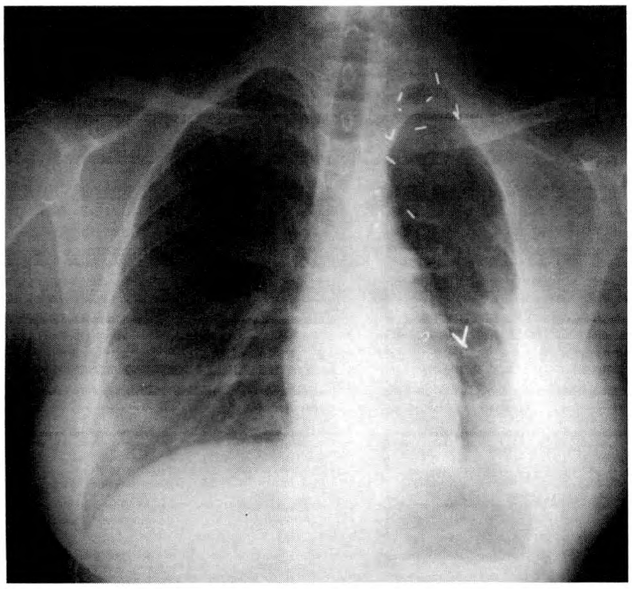

Fig 3. Postoperative chest roentgenogram shows complete reexpansion of the left lung and relief of mediastinal compression.

Clinical characteristics of 360 cases of benign mesothelioma have been reviewed [5]. There was a slightly higher incidence in females than males, the peak incidence was in the sixth and seventh decades of life, and the size of the tumors ranged from 1 cm to a massive tumor measuring 36 cm and weighing 4.54 kg. Although histologically benign, the overall mortality rate is about 12% owing to operative mortality, with removal or compression of mediastinal structures leading to fatal cardiopulmonary complications.

Grossly, these tumors appear as encapsulated, firm, and lobulated masses with a characteristic whorled appearance on cut surface. Eighty percent originated in the visceral pleura, and 20% originated in the parietal pleura. Interesting differences were noted between tumors larger than 8 cm and tumors smaller than 8 cm. The small tumors were more likely to be nodular within the lung, arising from the visceral pleura, and poorly vascular with no pleomorphism. On the other hand, the large tumors tended to be pedunculated within the pleural cavity, arising from the parietal pleura, and richly vascular with variable pleomorphism [5].

Treatment of benign localized mesothelioma consists of adequate local excision, removing only that portion of lung parenchyma necessary to ensure complete resection of the tumor. Great care must be taken at the time of operation because of the highly vascular pedicle. If the vascular nature of the pedicle, which can be awesome, is not appreciated, severe bleeding may occur. Apparent involvement of the chest wall is not necessarily a sign of malignancy or a contraindication for complete resection. Aggressive initial surgical excision and periodic long-term follow-up is necessary with these tumors. Local recurrence has been reported as long as 17 years after surgical excision [3].

We acknowledge the excellent assistance of Agatha Leighnor in manuscript preparation.

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Visceral and Limb Perfusion During Thoracoabdominal Aortic Aneurysm Repair

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Patients undergoing thoracoabdominal aortic aneurysm repair are at high risk of operative morbidity and death. Aortic clamping and unclamping stresses the myocardium, interrupts visceral and limb perfusion, and leads to metabolic acidosis. Use of a simple technique to preserve distal perfusion during the period of aortic clamping may reduce perioperative morbidity. We describe a technique of visceral and limb perfusion that may reduce surgical risk in high-risk patients.

(Ann Thorac Surg 1989;48:592–4)

Resection of a thoracoabdominal aortic aneurysm necessitates cross-clamping of the thoracic and abdominal aorta. Concerns have been expressed since the early development of thoracic aortic surgery over the interruption of visceral and distal limb blood flow [1]. Despite improvements in anesthetic management, aortic cross-clamping results in increased myocardial work and stresses cardiac reserve in patients whose cardiac status may already be compromised [2]. Plasma epinephrine levels increase significantly during aortic clamping, with further increases in myocardial work [3]. Aortic declamping produces a decrease in mean systemic pressure and may decrease coronary blood flow. Decreased coronary perfusion combined with metabolic acidosis may cause myocardial damage in patients with coronary artery disease and ventricular dysfunction.

Renal and visceral impairment after thoracoabdominal aortic operations are well-recognized complications [4, 5]. Carlson and colleagues [6] demonstrated that use of a shunt during thoracic aortic operations decreased the incidence of renal failure. Crawford and associates [7] concluded from their large experience that cardiopulmonary bypass was not necessary for thoracoabdominal aortic aneurysm repair.

In high-risk patients with severe coronary artery disease, ventricular dysfunction, and diffuse atherosclerosis, perfusion of viscera and limbs during thoracoabdominal aortic aneurysm repair may decrease perioperative morbidity and mortality. Distal continuous perfusion during the cross-clamp period may minimize changes in afterload and decrease cardiac stress. The incidence and extent of metabolic acidosis may also be decreased by maintenance of even limited perfusion.

Material and Methods

Patients

We describe the surgical results in 3 high-risk patients undergoing thoracoabdominal aortic aneurysm repair. All 3 patients had documented coronary artery disease and poor ventricular function (ejection fraction less than 40%). One patient had cardiogenic shock, myocardial infarction, and a ruptured thoracoabdominal aortic aneurysm and underwent an emergency operation. The second patient underwent an elective operation but was at additional high risk due to previous nephrectomy. The third patient had stable exertional angina and peripheral vascular disease and also underwent an elective operation.

Surgical Technique

Operative exposure was obtained through a left thoracoabdominal incision. Proximal and distal aortic control was obtained after radial division of the diaphragm and retroperitoneal exposure of the entire abdominal aorta. Both common iliac arteries were isolated. All patients were heparinized (1 mg/kg sodium heparin), and the uninvolved segment of the descending thoracic aorta (proximal to the intended site of clamping) was cannulated with a 6.5-mm Morris aortic cannula. A second cannula was placed in the abdominal aorta, proximal to the bifurcation. Both cannulas were interconnected through a manifold system containing individually controlled, self-inflating balloon catheters (Fig 1). The Morris cannula was unclamped just before clamping of the thoracic and abdominal aorta. The aneurysm was incised longitudinally to just below the level of the clamp. After removal of clots and atherosclerotic debris, balloon-tipped catheters were individually inserted to perfuse the celiac axis and the superior mesenteric and renal arteries (Fig 2).

All 3 patients received a HemoShield graft (24 mm). We used the inclusion technique with a circumferential running 3-0 Prolene (Ethicon) suture to attach a posterior elliptical opening in the graft to the visceral arterial orifices. The perfusion catheters were left in situ throughout the entire procedure and were brought out through the suture line at the thoracic aorta. The sutures were held

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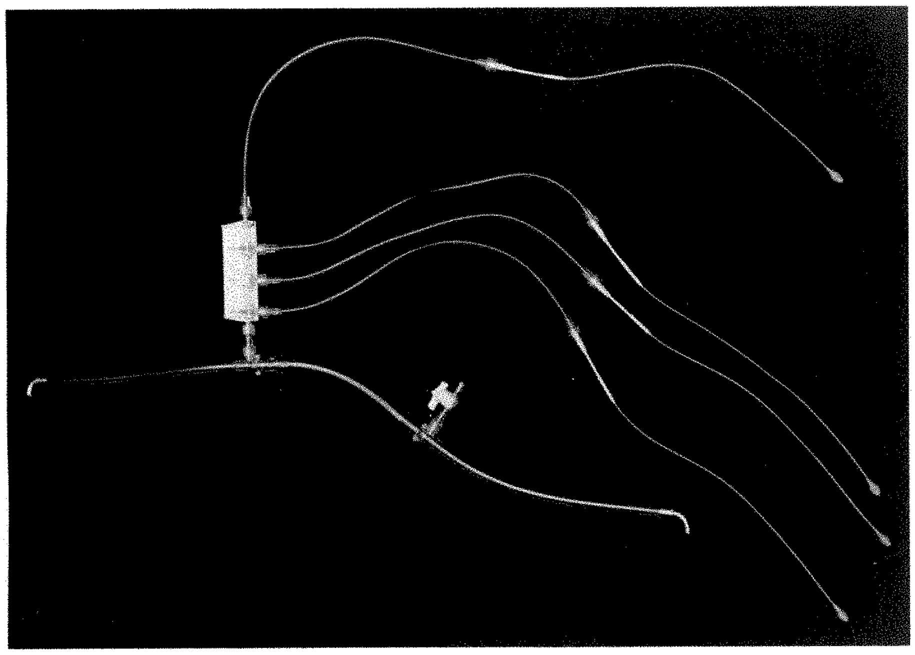

Fig 1. The catheter system used for shunting during thoracoabdominal aortic aneurysm repair includes two 6.5-mm Morris aortic cannulas interconnected by a manifold system with self-inflating balloon-tipped catheters.

under tension and were tied only after proximal and distal anastomoses had been completed and the cross-clamps and perfusion catheters had been removed.

Results

The patient who had a ruptured thoracoabdominal aortic aneurysm and myocardial infarction with cardiogenic shock died postoperatively due to extensive myocardial necrosis. The other 2 patients had an uneventful postoperative course, with extubation within 12 hours of operation and discharge from the hospital within ten days of operation.

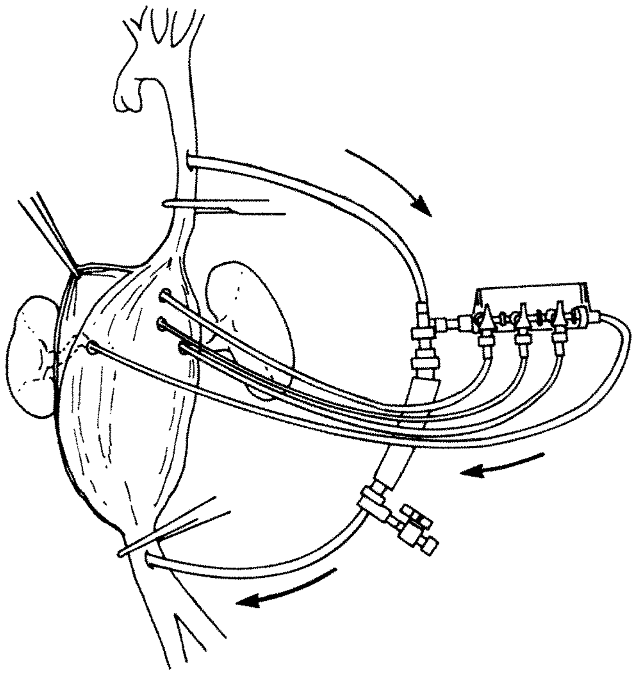

Fig 2. Technique of simultaneous visceral and limb perfusion during thoracoabdominal aortic aneurysm repair. Arrows *show direction of blood flow through the catheter system. Balloon-tipped catheters are inserted into the celiac axis and the superior mesenteric and renal arteries.*

None of the patients suffered from massive head and neck and upper limb edema, as is common after thoracic aortic clamping. The average cross-clamp time was 40 minutes. The average mean arterial pressure increased from 80 to 85 mm Hg after aortic clamping and decreased to 75 mm Hg with declamping. Nitroprusside was used in 2 of 3 patients but was discontinued at the time of cross-clamping. Positive inotropic agents were not required perioperatively except for the patient in cardiogenic shock preoperatively. Arterial blood gas levels were normal in all 3 patients five minutes after aortic declamping (carbon dioxide tension, 38 mm Hg; oxygen tension, 240 mm Hg; pH, 7.42). Urinary output was maintained at greater than 30 mL/h during the cross-clamping period, with no evidence of renal dysfunction postoperatively. The average blood loss was 1,240 ± 84 mL. Autotransfusion was used with the Haemonetics Cell-Saver system.

Comment

The operative mortality after thoracoabdominal aortic aneurysm repair varies between 10% and 50% [4]. The incidence of renal and visceral dysfunction is high even in centers with much clinical experience [4, 6]. We describe a technique of continuous visceral and limb perfusion during thoracoabdominal aortic aneurysm repair in high-risk patients. With autotransfusion, blood loss was minimal and surgical exposure was excellent.

Although we did not measure flow through the catheter system or distal vascular pressures, even limited perfusion is probably superior to no perfusion under normothermic conditions. Prolonged nitroprusside infusion during aortic clamping was not necessary and metabolic acidosis was not a problem after declamping.

In our experience, the maneuvers of aortic cannulation and perfusion did not add to the complexity of the operation. In this very small series of patients, we cannot provide scientific evidence of the effectiveness of this technique over that of the standard operation. We recommend, however, that the technique be considered in high-risk patients who have limited cardiac reserve and ventricular dysfunction. Further investigation with animal models and a larger surgical experience in high-risk patients will be necessary to provide validation and additional information.

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New Helper Instrument in Cardiac Surgery

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A new instrument has been designed for cardiac surgery. It frees the hands of the surgical assistant and is effective either for valve replacement or coronary graft anastomosis. The use of this instrument makes the operation quicker and easier.

(Ann Thorac Surg 1989;48:595–6)

A multipurpose malleable arm with adaptable accessories has been developed to fulfill some of the functions ordinarily assigned to the surgical assistant.* This device is easily attached to the cross piece of most sternal retractors. It is easily removable for convenience or sterilization. The other end of the device has a quick connector that receives three different accessories (Fig 1A).

The first accessory is a vise that securely holds the handle of all standard valve holders to facilitate passing sutures through the sewing ring. When a bioprosthetic valve is used, clips on the malleable arm can be used to hold a cannula that can drip saline or lactated Ringer's solution over the prosthesis and prevent drying (Fig 1B).

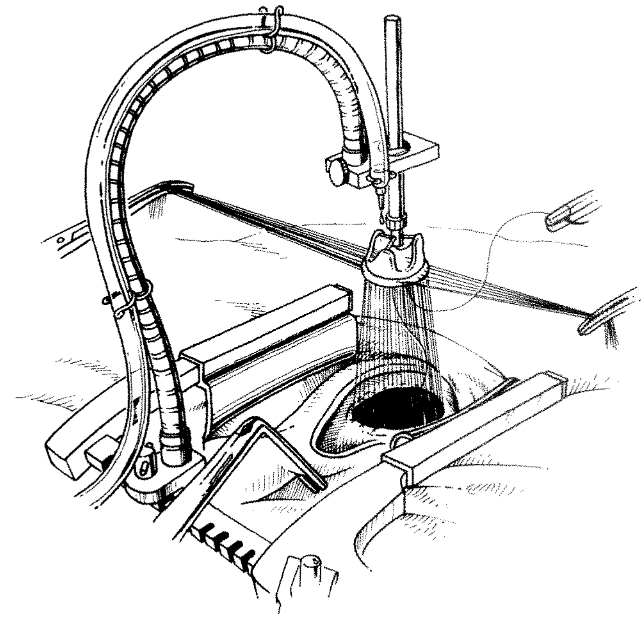

Fig 2. Aortic valve replacement with a bioprosthesis.

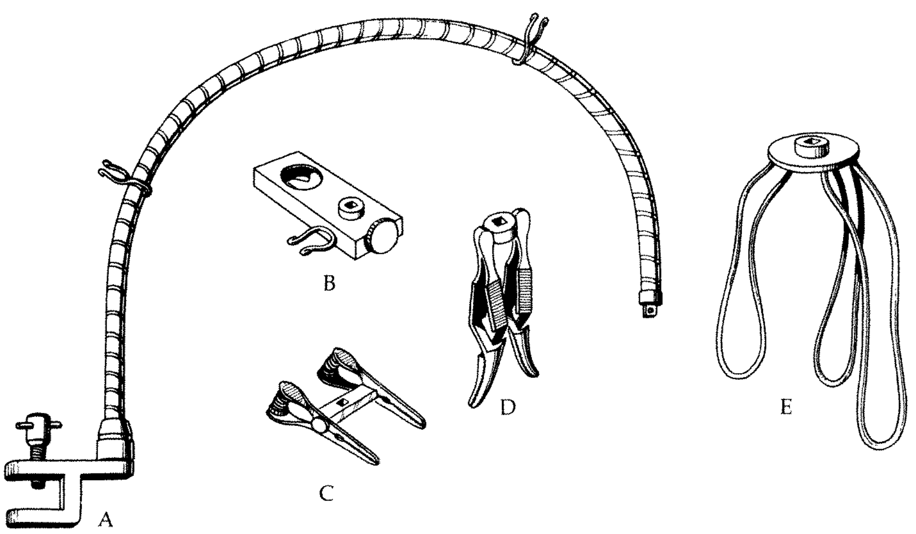

Fig 1. (A) Stainless steel malleable arm with a fixation system to a sternal retractor on one end and a quick connection for accessories on the other end. (B) Valve carrier vise. (C) Double bulldog atraumatic clamp for holding an internal mammary artery. (D) Double bulldog atraumatic clamp for holding the vein during sequential grafting. (E) Malleable atraumatic hand.

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* This instrument is patented and is manufactured by and commercially available through Delacroix Chevalier, Instruments de Chirurgie S.A., 13 Avenue de la Republique, 75011 Paris, France.

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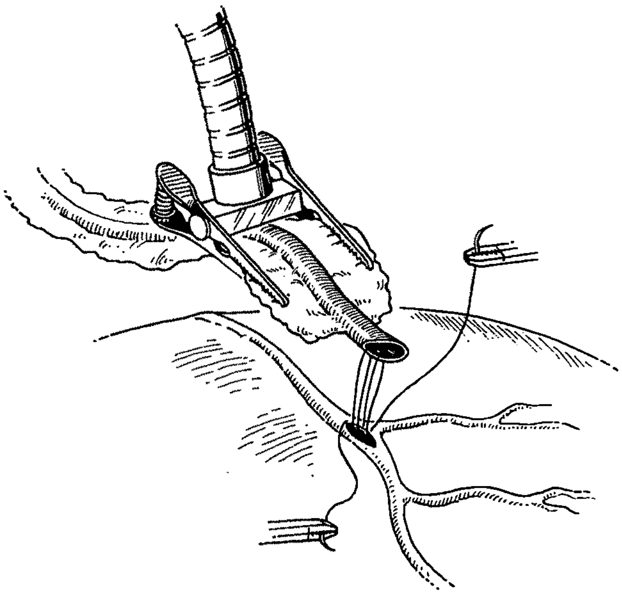

Fig 3. *Anastomosis of the internal mammary artery to the left anterior descending coronary artery.*

The second accessory has two bulldog atraumatic clamps mounted in a manner to allow holding the spatulated end of the internal mammary artery pedicle while sutures are being placed. It can also be used in a variety of situations during coronary artery bypass grafting (Figs 1C, 1D).

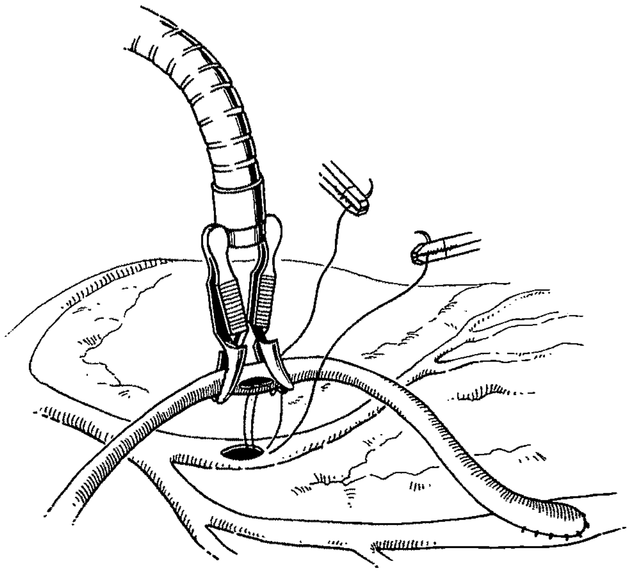

Fig 4. *Sequential coronary vein grafting.*

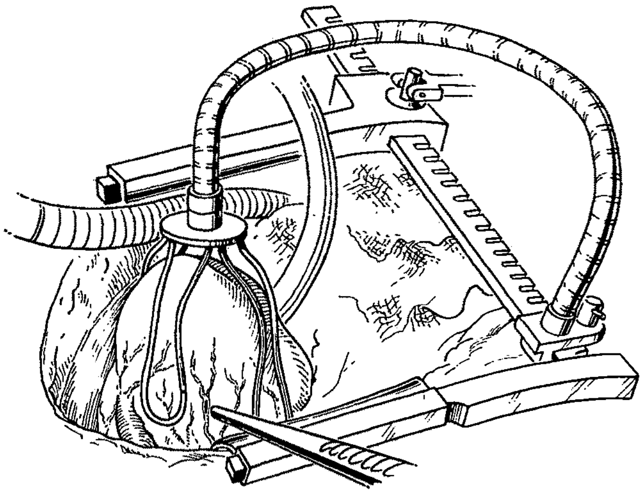

Fig 5. *The heart is elevated and rotated to the right side showing a coronary artery bypass graft on the obtuse marginal coronary artery.*

The third accessory is a malleable atraumatic hand, which can be used to support the heart and expose the lateral and inferior wall to facilitate coronary bypass grafting in these areas (Fig 1E).

Comment

This device has been used over a 6-month period in our department and has substantially facilitated valve replacement procedures (Fig 2). The device also assists the surgeon in coronary artery bypass grafting using the technique of continuous suture with the vein end or side opening held a short distance from the anastomosis before the sutures are snugged down (Figs 3, 4). The appliance for positioning the heart during exposure of the lateral and inferior wall has the further advantage of no warming of the heart by the assistant's hand (Fig 5).

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Gotthard Bülau and Closed Water-Seal Drainage for Empyema, 1875–1891

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Optimal treatment of pleural empyema remains controversial to the present day. In the preantibiotic era, surgical thinking favored early and aggressive drainage of closed-space infections, but the dynamics of the pleural space were poorly understood and open pneumothorax generally was considered the necessary price of surgical drainage. Against bitter opposition, revision of the dogma of early open drainage was achieved in 1918 by Evarts Graham and his associates on the US Army's Empyema Commission. Unacceptable mortality rates for early drainage were brought under control through a treatment program of repeated tapping, with surgical drainage only after loculation had occurred. Paradoxically, closed water-seal drainage for empyema had been used by a German internist, Gotthard Bülau, as early as 1875. His technique was published in 1891, 27 years before the report of the Empyema Commission. As a closed system, it would have been suited to empyema drainage in either the early diffuse or the loculated stages. Thoracotomy was not possible at the time, and Bülau probably could not foresee the future importance of his method to surgery.

(Ann Thorac Surg 1989;48:597–9)

The first therapeutic dilemma encountered by thoracic surgery in its infancy, suggested Dr Edward D. Churchill in a scholarly discussion, was the management of penetrating wounds of the chest [1]. Should the wound be closed to stop sucking and blowing of air into and out of the pleural space or left open to allow for drainage of blood, exudate, and the almost inevitable suppuration? "Close the wound," Ambroise Paré had concluded in 1575, on condition that blood was not retained within the chest, "for fear that cold air will penetrate in to the heart, and the vital spirits depart and vanish." In one guise or another, the dilemma of the chest wound has persisted almost to the present day.

Treatment of pleural empyema posed another intractable and recurring problem, one that reached crisis proportions during the world influenza pandemic of 1918 to 1919. The epidemic killed more people than did all the shells, trenches, machine guns, and poison gas of World War I. In the United States, draftees crowded into the Army camps were especially vulnerable to its complications. Churchill again describes the period vividly:

> The streptococcal pneumonia and empyema which accompanied the influenza epidemic was a new disease to a generation of doctors. They had no precedents which defined it. . . . Early evacuation of streptococcal pus was taught as a dogma of the surgery of the period. . . . Cyanotic patients, with pulmonary reserves crippled by massive and oftentimes bilateral bronchopneumonia, were being hurried to an operating room as soon as thoracentesis yielded fluid containing chains of streptococci. The operation was rib resection with open tube drainage. Death occurred quite frequently about half an hour after the operation [1].

A survey by the Surgeon General of the Army in February 1918 found mortality rates for empyema drainage in the base hospitals to be averaging 30% [2]. He appointed a commission to study the problem, headed by Major Evarts A. Graham; the Commission's conclusion and recommendations are a familiar landmark:

> In the light of all these observations it would seem definitely established that an operation for empyema performed too early in the course of the disease is accompanied by such very grave danger that in our opinion the risk of harm by the operation outweighs any advantages which it may have. . . . The only advantage to be gained from an early operation is drainage which theoretically accomplishes the removal of both toxic material and living organisms and in addition relieves mechanical embarrassment to respiration caused by the presence of a large amount of fluid. Practically, however, the mechanical embarrassment to respiration is almost sure to be aggravated instead of improved because of the creation of an open pneumothorax; and moreover, the fluid can be withdrawn by aspiration as often as it accumulates in any considerable amount, a procedure which, of course, also removes some of the toxic material and living organisms. *The apparently theoretically ideal method of continuous drainage under negative pressure involves the necessity of a special attendant to prevent a delirious patient from interfering with the apparatus and thus running the risk of creating an open pneumothorax* . . . [Italics added].
>
> Practically it may be stated that, in general, the safest time to operate is when the exudate has become frank pus instead of being merely serofibrinous, for the following reasons: 1) There is less danger of creating an open pneumothorax because, in our experience, there is likely to be a circumscribed abscess shut off by adhesions from any communication with the free pleural cavity so that during the operation the pleural cavity, properly speaking, is not entered . . . [2].

Immediately after adoption of these changes, mortality of empyema drainage in Army hospitals decreased to 4.3% [2]. Closed catheter drainage obviously was known

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Quotations from Bülau's paper are in translation by Dr Meyer.

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Fig 1. Gotthard Bülau (1836–1900) of Hamburg, Germany, originator of the method of closed water-seal drainage of the chest. (Reprinted by permission of the publisher from Nissen R, Wilson RHL. Pages in the history of chest surgery. Springfield, IL: Charles C Thomas, 1960.)

in the United States at the time, but was not entirely accepted as a reliable method of treatment. Why not? The reasons may be difficult to understand after this lapse of time. Graham and Bell [2] perceived the risk of the patient accidentally pulling out the tube. Tube drainage may have been regarded as a method developed in the enemy country; Major Pierre Duval of the French Army medical corps and Colonel Berkeley Moynihan of the British publicly condemned German surgical methods (such as differential pressure chambers) during lecture tours of the US [1]. And finally, surgery in the US was barely arriving at the point at which the open chest under anesthesia could be considered even a possibility. Physiological findings from the laboratory had not yet made much impression on surgical practice. Suction drainage of the pleural cavity after lobectomy would not be described for more than a decade, in the report of Harold Brunn [3]. Closed water-seal drainage for empyema, however, dated back at least to 1875.

A Prophetic Insight

Gotthard Bülau (Fig 1), born in Hamburg in 1836, practiced internal medicine with an interest in pulmonary disease. Thoracic empyema, usually postpneumonic, was an unsolved problem. Surgical treatment in the acute phase consisted of rib resection with open tube drainage; in the chronic phase, the thick-walled indurated space was often managed by excision of the overlying ribs and chest wall, "saucerization" of the space, as it came to be called. Surgeons for the most part considered postoperative deaths to be the result of infection rather than a superimposed respiratory handicap. Few accepted the validity of laboratory studies showing the importance of negative intrathoracic pressure. Bülau was familiar with these concepts, however, and evolved a method of closed drainage which he eventually published in 1891 [4].

> In the proceedings of this year's medical congress in Vienna on the management of empyema, only two methods came under discussion: the radical operation involving resection of the overlying ribs, and siphon-drainage. While advocates of the first method were in the majority,* the last-named method also had its strong supporters, and the results they presented deserve comparison. Although the results obtained recently after rib resections have been extraordinarily favorable, the question remains whether in the majority of cases, one would not wish to use a safer and less complicated method. There can be no difference of opinion, that siphon drainage involves a less severe operative injury. . . . *I have always believed that the principal advantage of siphon-drainage is that it lowers the pressure within the pleural space, thereby bringing about reexpansion of the lung* [Italics added].

Bülau described his technique of drainage in detail (p 37):

> It is easier on the patient, if one first makes a small incision through the skin with a Bistoury or lancet, over the site where the trocar is to be inserted, long enough to allow the trocar to pass through; the skin itself offers the principal resistance, and if not incised will make the trocar puncture difficult and painful. Immediately after removal of the trocar-point, the catheter is passed through the cannula, the cannula is pulled back over the catheter, and the skin is securely closed around it. Then, it is convenient to clamp the catheter for a short time and to secure it in place, before connecting it to the siphon apparatus. I place a suture in the skin, tying it directly to a safety pin placed in the catheter at the level of the skin; both ends are secured, and the site is covered with gauze sponges and collodion, or by strips of adhesive plaster extending from front to back of the chest wall . . . After the catheter has been secured, it is attached to the siphon-apparatus which has previously been filled with antiseptic solution. The free, weighted end of the rubber drainage tubing is immersed in a bottle filled one-third full of the same solution, and the occluding clamp is removed. Now the pus flows slowly out of the chest cavity, and as a result the lung must expand by a precisely equal volume.
>
> A precautionary measure, to which little attention has been paid, is to secure the drainage tubing to the edge of the patient's bed or mattress, and indeed to allow a slack loop of 40 to 50 cm of the tubing to lie beside the patient in bed. By doing this, one can prevent the weight of the tubing and apparatus from pulling constantly on the catheter within the chest, thereby causing pain and the risk of its slipping out; also it allows the patient free movement in bed and the ability to turn into a comfortable position . . . If the apparatus functions well from the beginning, and the pus is completely evacuated, we can expect the fever to fall immediately in uncomplicated cases. If the fever recurs, suppuration in the drainage tract is usually to blame, or else the catheter has become plugged by some kind of obstruction.

Bülau reported several case histories, the earliest that of a man treated in 1875 (p 41).

> W., 35-year-old carpenter, admitted April 11, 1875. Illness began with chills on April 6th. Pleuropneumonia left lower lung. High fever, delirium. Beginning on the 17th, profound

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* [Present author's note] Bülau mentions Schede several times as having been present and among the majority favoring resection of the overlying chest wall.

> sweats with intermittent fever. At the beginning of May, dullness over the entire left chest, which had 3 cm greater circumference than the right. Displacement of the heart. Edema of the legs and the left chest wall. May 4: 2,250 cm^3 of pus withdrawn by tapping and siphon-drainage. May 17: daily drainage of about 100 cm^3 of pus; catheter easily obstructed by blood clots. Injection of calcium solution. Patient's general appearance much better. Continued improvement; discharged September 6 with the fistula closing.
>
> Readmitted June 28, 1876. Two months after discharge from the hospital, exudate again had appeared from the drain wound, with alternate coughing and purulent discharge. Examination of the chest showed no significant differences between the two lungs. Catheter was again placed into the fistula and connected to siphon drainage. Thereafter, cough ceased and purulent drainage was minimal. July 25: the patient was discharged with drain in place, and returned every 8 days for dressing. In November the drain was removed, with subsequent complete healing. Over the intervening years, I have observed him to maintain full health and activity.
>
> [Concluding, p 45] In view of the observation reported here, of healing of an empyema fistula of 15 months' duration under siphon-drainage, with reexpansion of the lung, I believe that if faced with a similar case one would be entirely wrong not to make a search of the literature for alternative treatment, before proceeding to chest-wall resection.

Did Bülau foresee the importance of his method to the future development of surgery? He speaks of the method as siphon-drainage, implying that he considered it a hydraulic mechanism for evacuation of fluid from the chest. Did he understand also that closed water-seal drainage could bring about reexpansion of the lung (assuming no air leaks from the lung) solely on the basis of the patient's own respiratory movements?

> In this situation the drainage can function as a valve, allowing escape of pus and the air which has entered during the operation, while preventing renewed entry of air from without . . . With each forcible expiration against a closed glottis, the air in the opposite (healthy) lung is forced into the partially collapsed one, inflating it and driving out a corresponding amount of drainage through the cannula. With the next inspiration the valve closes, the lungs are further expanded, the expansion is maintained, and with the next expiratory stroke expansion proceeds a step further [4].

Closed drainage: could it be used for procedures other than empyema drainage? In 1891 when the article was published, intentional thoracotomy was not possible in any case. No method existed for maintenance of pulmonary ventilation under anesthesia in the presence of open pneumothorax. The chambers incorporating differential pressures, Unterdruck and Überdruck, were not to be described until 13 years later, in 1904 [5, 6]. Thoracotomy had not yet been developed and Bülau was not a surgeon, but his insight would be indispensable to the surgery of the future.

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Allograft Aortic Valve Implantation: Techniques for All Types of Aortic Valve and Root Pathology

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The geometry and degree of symmetry of the diseased aortic root and valve dictate the technical method of implantation of the allograft aortic valve. Five methods are available that are suitable for the full range of aortic root disease: the small aortic root with a valve annulus diameter less than 21 mm, the common aortic valve lesions (valve annulus diameter, 21 to 29 mm), the aneurysmal noncoronary sinus, the moderately large annulus (valve annulus diameter greater than 30 mm), and the aneurysmal aortic root and dilated annulus. Implantation methods include the subcoronary technique, miniroot inclusion technique, and aortic root replacement. Technical variations such as valve inversion during implantation, valve rotation, and continuous or interrupted suture methods are important in certain techniques. The allograft aortic valve is a versatile device that can be used in the surgical management of the full range of aortic valve and aortic root pathology.

(Ann Thorac Surg 1989;48:600–9)

As a consequence of the encouraging long-term results of the allograft aortic valve in the subcoronary position [1, 2], many cardiac surgeons have developed an interest in adding this technique to their surgical armamentarium. However, the surgeon is confronted by an array of potentially confusing technical variations such as valve inversion during implantation, valve rotation, continuous or interrupted suture technique, aortic root tailoring, aortic annulus enlargement, partial preservation of allograft aortic sinus wall, and aortic root replacement [3–6].

The allograft aortic valve is a very versatile device that can be used in the management of the full range of aortic valve and aortic root pathology. For instance, with the large host aortic annulus (30 mm or greater), the incidence of valve incompetence after subcoronary implantation and aortic root tailoring is higher than that for the smaller host annulus [7, 8]. Currently an allograft aortic valve implantation technique to obviate aortic root tailoring is available. Furthermore, aortic root replacement by an allograft is an important alternative to the mechanical or bioprosthetic valved conduit.

Because anticoagulation can be avoided and because of a very low incidence of valve failure, endocarditis, and thromboembolism [9], the allograft aortic valve is indicated in virtually all patients with aortic valve and aortic root pathology, with the possible exception of the elderly patient in whom a xenograft aortic valve would give a comparable result. Consequently, it is necessary for the surgeon training in the use of the allograft valve to develop the flexibility to apply the specific technique to the particular aortic pathology.

It is timely to review the aortic root anatomy and pathology as they relate to valve replacement, as well as the technique of subcoronary implantation of the allograft aortic valve, in particular addressing the place of each of the technical variations. The allograft miniroot inclusion and aortic root replacement techniques will be outlined, with special reference to their use for the appropriate aortic root pathology.

The methods that will be described are in part based on the 20-year experience with allograft valves at The Prince Charles Hospital.

Aortic Root

The aortic root and valve are an integral unit and should be visualized spatially as such. The normal structure is essentially a symmetrical cylindrical tube and, with this concept in mind, insertion of a competent allograft aortic valve can be achieved.

Pathological lesions of the aortic root present variable architecture ranging from perfect symmetry to gross asymmetry and disorganization, as in some cases of staphylococcal aortic root infection. For the purposes of deciding the appropriate surgical procedure, it is convenient to categorize the aortic root and annulus on the basis of two measurements, the valve annulus diameter (VAD) and the sinotubular or transcommissural diameter (STD) (Fig 1A). In the normal aortic root, the STD is usually 2 to 3 mm larger than the VAD. These measurements and the overall configuration of the root can be calculated preoperatively from the cineaortogram if a measuring grid is incorporated in the film. This is the method that we prefer. The width of the contrast in the immediate subvalvar region on a left ventricular injection or reflux of contrast from an aortic root injection in the presence of aortic regurgitation can also provide a reasonably accurate measurement of the VAD (Fig 1B). Echocardiography is helpful but experience at The Prince Charles Hospital has shown that the VAD estimations, especially with a calcific immobile valve, may be inaccurate. The purposes of these

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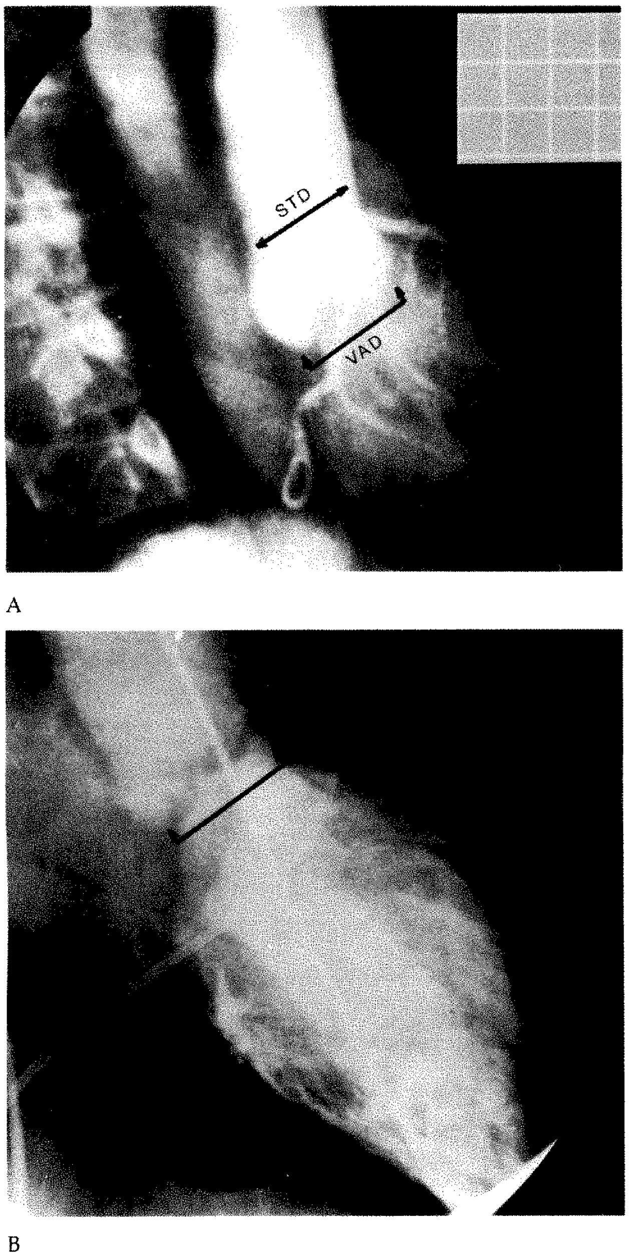

Fig 1. (A) Cineaortogram demonstrating the valve annulus diameter (VAD) and sinotubular diameter (STD). A 1-cm measuring grid has been incorporated in the film. (B) Left ventriculogram demonstrating the immediate subaortic valve dimension, which approximates the VAD.

measurements are to give the surgeon an appreciation of the aortic root symmetry and to select a range of appropriate-sized valves to be brought to the operating room. With experience, these measurements may become superfluous.

Lack of aortic root symmetry with a substantial discrepancy between the VAD and STD is often seen in aortic root pathology. The STD is greater than the VAD in poststenotic dilatation and in aneurysmal aortic root disease (Fig 2A). Occasionally, removal of a stenotic bicuspid aortic valve results in the VAD being greater than the STD (Fig 2B). In situations in which the noncoronary sinus alone is aneurysmal, or at least considerably larger than the other two sinuses, the VAD and STD may not be parallel (Fig 2C).

In most instances of a calcified bicuspid aortic valve, there is an anteroposterior slitlike orifice, a heavily calcified fused anterior commissure, and a left lateral rudimentary raphe. After valve excision, the three sinuses are virtually equal and little problem exists with the insertion of a symmetrical leaflet valve. When the bicuspid valve has a transverse orifice, however, two sinuses generally exist and the two coronary ostia may be directly opposite. This requires special care to ensure that none of the commissural pillars of the trileaflet allograft valve impinge upon a coronary ostium.

Allograft Aortic Valve

The methods of allograft valve procurement, trimming, and cryopreservation at The Prince Charles Hospital have been previously outlined in detail [10, 11]. In summary, valves are harvested from organ donors and from coroner's autopsies within 24 hours of death. Donors must be less than 55 years of age and without evidence of systemic infection or cancer. The heart and great vessels are removed in a sterile manner and transported to The Prince Charles Hospital. The aortic and pulmonary valves are separated and excess tissue is excised. Of paramount importance at all stages of valve handling, from harvesting to implantation, is observance of a "no touch" technique of the leaflet tissue. It is believed that failure to observe this principle is injurious to the leaflet tissue, and may adversely affect long-term valve durability. The internal orifice is measured by clamping the distal aorta and passing a feeding tube into the aorta via one of the coronary arteries. This tube, when inside the aorta, is fixed in the distal aortic clamp. Both coronary arteries are ligated, the valve is inverted, and the root is distended with M199 nutrient medium with antibiotics (cell culture medium [Commonwealth Serum Laboratories, Melbourne, Australia] with penicillin [50 IU/mL] and streptomycin [50 μg/mL]). The diameter of the valve annulus is measured with calipers and the competence of the aortic valve is checked. Clamping the tube within the distal aortic wall clamp prevents its tip from damaging the leaflet tissue. After sizing, valves obtained at coroner's autopsies are incubated at 37°C in nutrient medium with antibiotics for six hours and then cryopreserved, generally within 21 hours of donor death. Valves from organ donors are cryopreserved immediately after sizing, generally within four to six hours of donor death.

The aortic and pulmonary valves are cryopreserved by sealing the valves in separate bags each containing 90 mL of cold (4°C) M199 without antibiotics and 10 mL of 10% dimethylsulfoxide. Cryopreservation at a rate of −1°C per minute in liquid nitrogen using a controlled-rate freezer to −40°C is carried out before transferring the valve to a liquid nitrogen storage tank for long-term storage at approximately −196°C.

Extensive cultures of all processing solutions and valve trimmings are performed at all steps as surveillance for

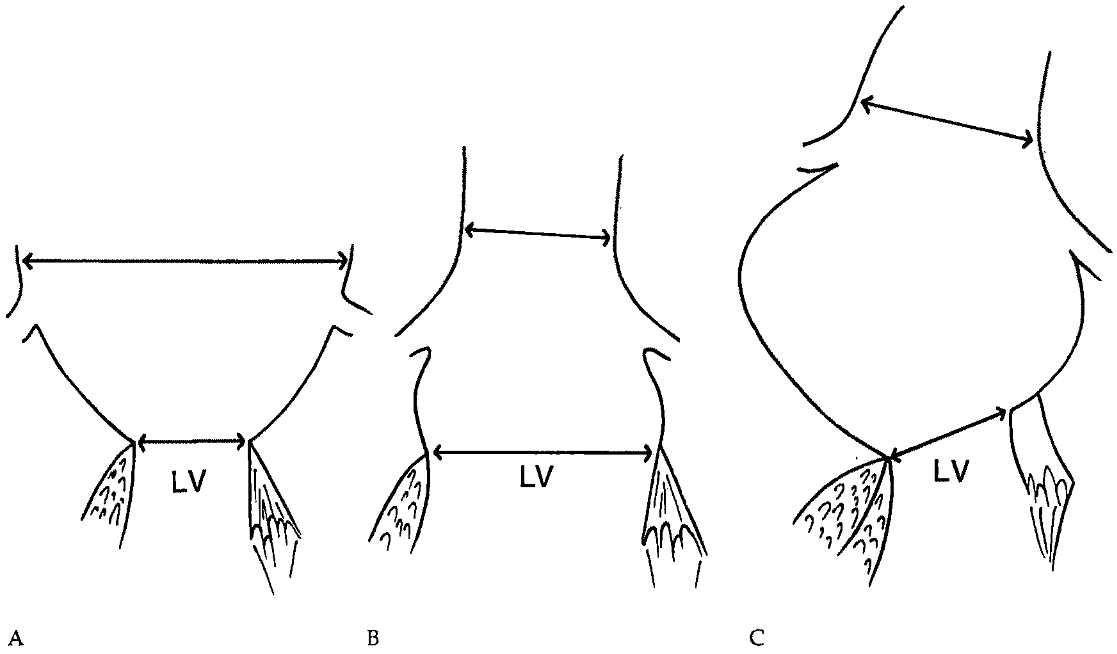

Fig 2. (A) Sinotubular transcommissural diameter greater than VAD: as in poststenotic dilatation and in aneurysmal aortic root disease. (B) Valve annulus diameter greater than STD: occasionally seen after removal of stenotic bicuspid aortic valve. (C) Sinotubular transcommissural diameter and VAD not parallel: seen where noncoronary sinus is aneurysmal. (LV = *left ventricle.)*

microbiological contamination. Valves are not released for use until the results of all cultures at 3 weeks are negative.

Valve leaflet viability is inferred from the demonstration of a positive tissue culture from a piece of tricuspid valve that had undergone the same cryopreservation process.

Technique of Aortic Valve Replacement With an Allograft Valve

The usual technique of allograft aortic valve replacement applies to the most common valve annulus diameter, that is, between 22 and 29 mm. Alternative techniques are required for patients with asymmetry of the aortic root, valve annulus diameters outside this range, and aortic root aneurysmal disease. Each of these five methods will be described.

The operation is performed through a median sternotomy employing the usual techniques of cardiopulmonary bypass and myocardial protection with cardioplegia every 20 to 30 minutes supplemented with pericardial ice slush and intracavity cold Ringer's solution, especially for the hypertrophied left ventricle. The left side is vented by a left apical ventricular or atrial catheter. A vertical aortotomy incision is made curving down into the noncoronary sinus. Stay sutures are placed on the edge of the aortic incision.

The presence of coronary ostial abnormalities (early division or double-orifice left coronary artery), the size of the ventricular membranous septum (and therefore the proximity of the valve annulus to the conducting bundle), and the pathology of the aortic valve are determined. An appreciation of the symmetry of the aortic root is gained at this stage as well. The valve is excised, taking care to aspirate all loose pieces of calcium. The valve orifice can often be assessed visually but is always measured with any of the standard valve obturators. It is important to choose the sizer that will just rest within the annulus, inspecting gently for any possible space.

During the thawing of the allograft, the surgeon can perform any concomitant procedure such as coronary artery bypass grafting. Decalcification of the aortic root can be completed and the initial three sutures for the subcoronary implantation can be inserted.

Valve Thawing

If the valve supply is satisfactory, two or three valve sizes are transported to the operating room in a portable liquid nitrogen cylinder. The selected valve is removed from its outer bag and, while still in its inner bag with a piece of tricuspid valve, is thawed rapidly in a sterile water bath at 40°C. After approximately four minutes, the tissues are removed from this bag and rinsed stepwise for two minutes each in each of four bowls: the first contains 500 mL of M199 with 5% dimethylsulfoxide, the second contains M199 with 2.5% dimethylsulfoxide, and the last two contain M199 but without dimethylsulfoxide at 4°C. The valve is handed to the surgeon for examination and further trimming.

Size of Allograft Valve

The combined thickness of opposite sides of the allograft valve at its base is approximately 3 to 4 mm. The internal diameter of the selected allograft therefore should be 3 to 4 mm less than the measured host VAD. These guidelines produce a competent valve for the lower VAD range. The technical errors of implantation are more likely to occur as the host VAD increases. Therefore, with the larger annulus diameter, an allograft valve with an internal diameter

2 to 3 mm less than the host annular diameter will be more likely to produce ideal leaflet coaption and avoid leaflet prolapse.

Type of Suture Material

The ideal needle is a strong taper-cut V-5 half-circle on either a 3-0 polypropylene or a 3-0 green braided polyester Ethibond B 936 suture. Polypropylene is preferred, but the needle in certain situations is more important than the suture material. Such a half-circle needle facilitates suturing through tough tissue at difficult angles. Obviously any interrupted suturing should be with polypropylene, allowing the tissue to glide as the valve is guided into position. For the small annulus where access may not be easy, the strong half-circle needle is superior. For the large annulus, where visualization is not a problem, 4-0 Prolene on a large (Taper SH-8521, Ethicon) and a small (Taper RB-8557, Ethicon) needle can be used for various parts of the operation.

Leaflet Coaption

It is possible that the normal human aortic valve leaflet coaption during diastole is as much as 40% of the total ventricular surface of each leaflet. Probably, the most common cause of early postoperative valve incompetence is leaflet prolapse with inadequate coaption. Various technical maneuvers are described to produce symmetrical implantation.

These methods correctly align the valve in a horizontal plane, but equally important is the height of fixation of the commissural pillars in the vertical plane. As a general and important rule, the top of the allograft commissural pillar, which includes 3 mm of aortic wall above the top of the leaflet, should be 10 mm cranial or distal to the top of the host commissure leaflet. Coaption will be maximal if this rule is observed, provided that the STD approximates the VAD and the correct size of allograft has been chosen.

When to Invert

Inverting the allograft into the left ventricular cavity to perform the continuous proximal suture line not only apposes endocardial tissue of both graft and host but simplifies the suturing of these layers. The only disadvantage is that the allograft is doubled back on itself, increasing the tissue bulk in the outflow tract. Therefore it is not advisable in the small root (VAD less than 21 mm).

When to Rotate

Rotation of the allograft 120 degrees anticlockwise brings the allograft right coronary sinus and annulus against the host left coronary sinus. The advantage of rotation is to bring the allograft proximal muscle rim posteriorly, where its bulk protrudes less into the left ventricular outflow tract. Rotation is performed in most subcoronary implants, but should be avoided in two situations. First, sometimes one of the sinuses of the allograft is smaller than the other two. If the host has similar unequal sinuses, the allograft can be oriented accordingly, often without rotation, placing the small allograft sinus against the small host sinus.

Second, for the aneurysmal noncoronary aortic sinus, the allograft noncoronary sinus wall is left intact and no rotation is performed. This technique is the standard procedure by Ross and associates [12] for most subcoronary implants.

Valve Trimming

The muscle beneath the base of the valve is carefully trimmed down to a thickness of 3 mm. Where a small allograft (18 mm or less in internal diameter) is being used, the valve should be thinned more than for a larger valve to avoid obstruction caused by the bulk of allograft valve tissue in a small valve orifice. Either at this stage or later, the valve pillars supporting the commissures are fashioned by excising the intervening aortic wall in a U shape. This leaves a rim of aorta that is approximately 3 mm wide around the base of each leaflet (Fig 3A).

Technique of Implantation (Valve Annulus Diameter, 22 to 29 mm)

The valve is turned inside out and inverted, and the three double-armed 3-0 braided polyester sutures are then passed in turn through the allograft at the base of each cusp. The equidistant placement of these three sutures in both the host annulus and the graft is considered a most important technical step. The symmetrical insertion of the valve is dependent on the accuracy of placement of these three sutures (Fig 3B).

The allograft is lowered into the left ventricular outflow tract and each suture is tied (Fig 3C). The proximal suture line is begun using one arm of each of the braided polyester sutures (Fig 3D) in a continuous over-and-over technique. The important feature of this suture line is that it does not follow the scallops of the host annulus but proceeds in a horizontal line beneath the commissures. The only exception to this is in the region of the membranous septum, where the suture line should be through or just below the annulus to avoid injury to the conduction system, which is especially likely to occur when the membranous septum is small, the bundle being consequently closer to the annulus. The allograft valve is brought into normal orientation by drawing the pillars up into the aortic root and then placing a stay suture at the top of each pillar (Fig 3E). The distal suture line is performed using three similar double-armed 3-0 braided polyester sutures. Taking the knots of the previous three sutures of the proximal suture line as the guide, each suture of this distal suture line is placed exactly cephalad to this knot. The suture is passed through the allograft aortic wall at the center and deepest point of each sinus and then through the adjacent host aortic sinus wall just distal to the knot (Fig 3F). Therefore, if the first three equidistant sutures of the horizontal layer are correct, then these last three sutures of the distal suture line will also be correct. After tying these three sutures, the distal line is then begun at the noncoronary sinus. One arm of the suture proceeds with an over-and-over technique along the posterior half of this sinus up to the top of the pillar. The limb of the left coronary suture is then taken along the right side of this sinus to the top of the posterior pillar. The two sutures at the top are then brought

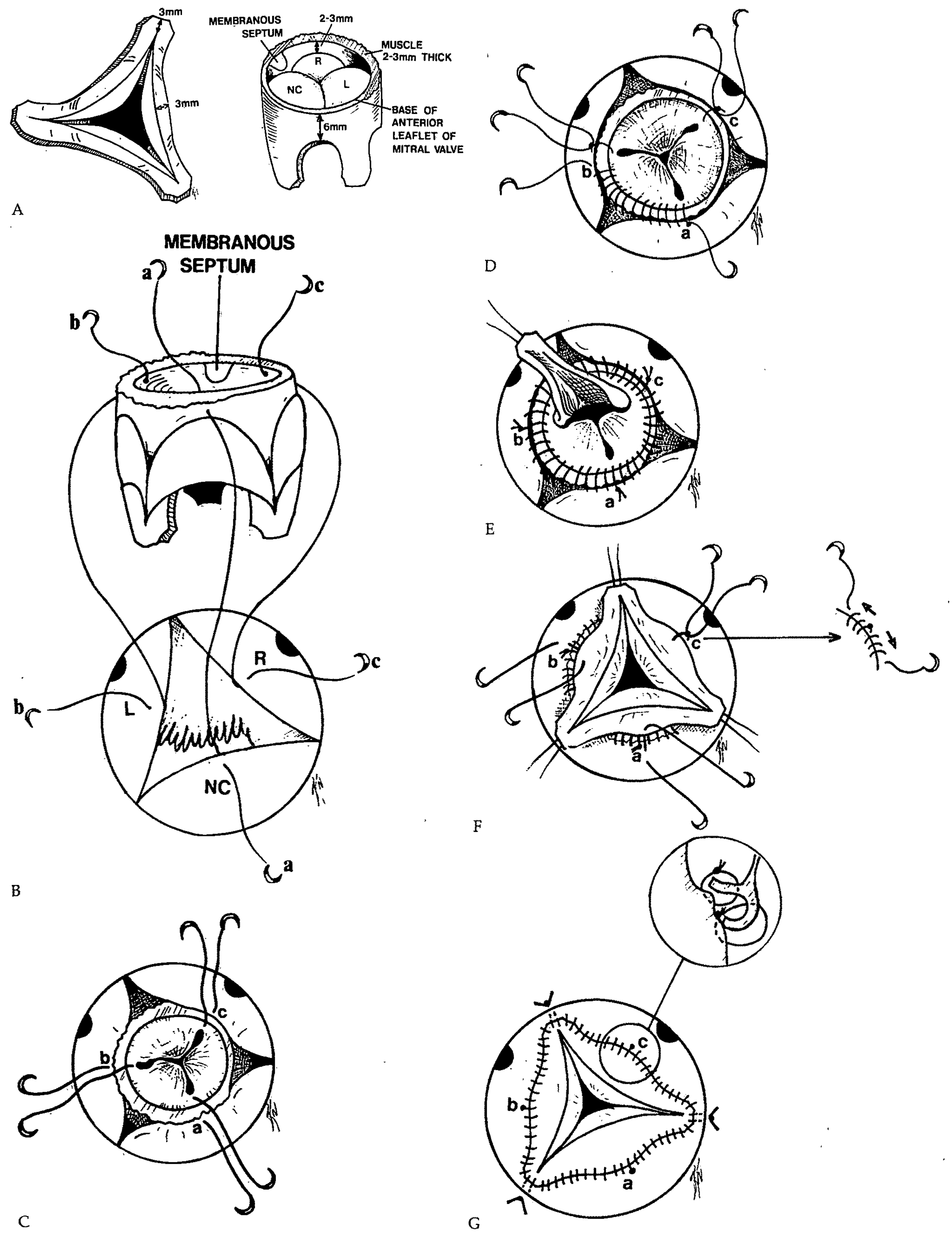

Fig 3. (A) Appearance and dimensions of the allograft after trimming. (B) The valve is turned inside out and then inverted. The three sutures are placed for the proximal suture line. (C) The valve is lowered into the left ventricular outflow tract. (D) The proximal suture line is performed. (E) The pillars of the valve are brought up into the aortic root. Stay sutures are attached to the adventitia of each pillar. (F) Sutures for the distal suture line are placed. (G) The distal suture line is performed, beginning in the noncoronary sinus. (L = *left;* NC = *noncoronary;* R = *right.)*

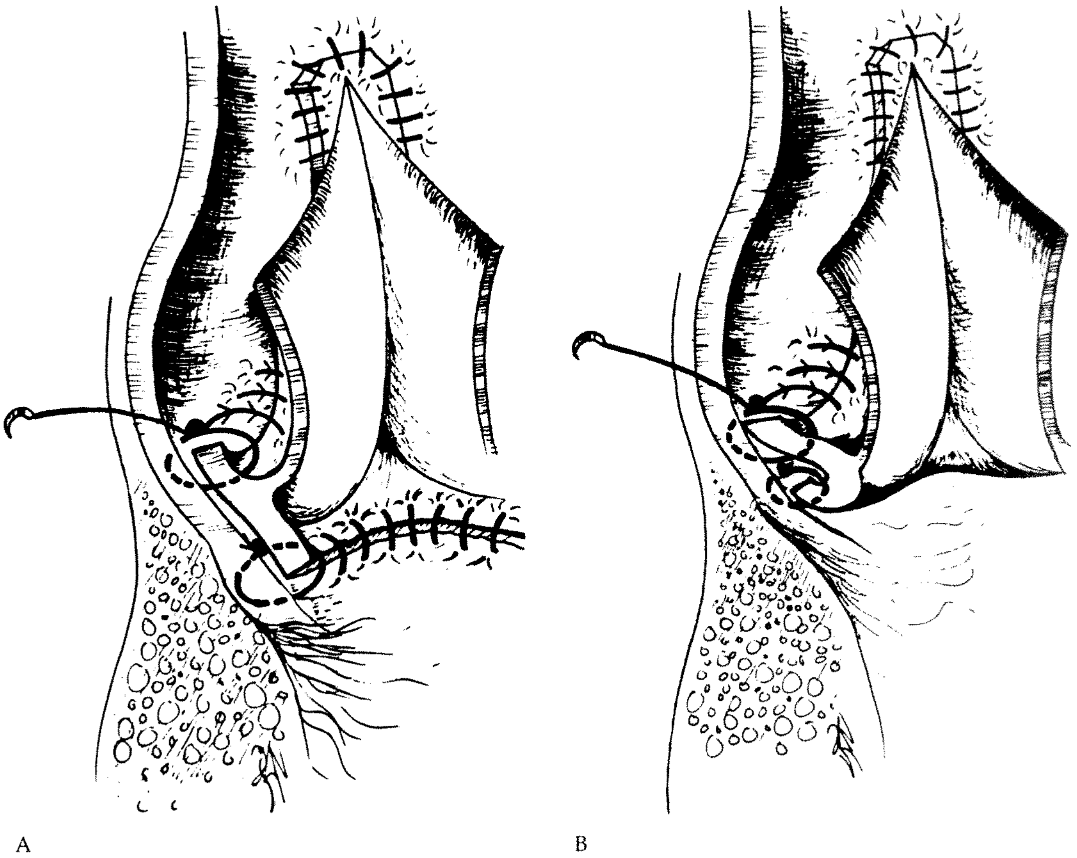

Fig 4. The small aortic root. (A) Interrupted proximal suture line without inversion. (B) Continuous proximal suture line with inversion, which may result in excessive allograft muscle bulk.

through the pillar, through the host aortic wall, and tied outside the aorta without Dacron felt. The other two pillars are sutured in a similar manner (Fig 3G). Each pillar is fixed to the host aortic wall about 1 cm above the top of the host commissural leaflets.

In some instances, if one arm of the suture is completed to the top of a pillar, the visualization of the corresponding arm may be more difficult. It is sometimes easier to suture half way on one side and then go to the adjacent suture to complete half of the other side of the same pillar. The height of the pillars 1 cm above the host commissure needs to be judged carefully and their position correctly sited in both the vertical and horizontal planes, particularly the two pillars on each side of the aortotomy incision. The distance between these pillars should be a little greater than the distance between the other pillars to allow for the aortotomy closure. On most occasions at least two of the host commissures serve as vertical markers over which the allograft pillars will be sutured. If the STD is greater than the VAD, the excess is often corrected by a generous closure of the aortic incision. If it is too small by some 3 to 4 mm in diameter, then a pericardial patch is indicated; it is inserted before the distal suture line of the allograft is begun and before the allograft pillars are sited.

Special Situations

Small Aortic Root (Valve Annulus Diameter Less Than 21 mm)

This small annulus is seen more frequently in elderly women and in children with congenital valve stenosis. The valve is bicuspid or rarely unicuspid (replacement being required after a previous valvotomy). Less commonly seen is tunnel aortic stenosis with marked obstruction of the left ventricular outflow tract, which may require aortic root replacement [13] or aortoventriculoplasty [14] techniques combined with aortic valve replacement.

A small annulus of 19-mm diameter in an active patient whose body surface area is greater than 1.5 m^2 is enlarged by a pericardial or Dacron gusset [15, 16]. A 7-mm-wide patch (after suturing) enlarges the VAD from 19 to 21 mm, enabling an allograft valve of 18-mm internal diameter to be inserted. In smaller patients annular enlargement may not be necessary as an allograft with an internal diameter of 16 mm can be inserted into a 19-mm host root without a clinically significant gradient [17].

The technical modifications for the small aortic root are (1) to avoid inverting the allograft while performing the proximal suture line and (2) to insert simple interrupted

4-0 Prolene sutures. Without inversion of the valve excessive allograft tissue bulk in the left ventricular outflow tract is avoided (Figs 4A, B). Likewise, continuous suturing is not feasible in the small root as the valve needs to be held out of the aortic root to provide exposure. Although rotating the valve 120 degrees to the left is not essential, it does bring the allograft muscle bulk posteriorly with less turbulence possibly than in the anterior position.

The distal continuous suture line is the same as described for the usual subcoronary technique. The selected allograft should have an internal diameter 3 mm less than the annular diameter of the host. A 4-mm difference should still produce a competent valve.

Large Asymmetrical Noncoronary Sinus

When the noncoronary leaflet is larger than the other two leaflets, its coronary sinus is often aneurysmal. This anatomical anomaly presents difficulty with the usual implantation technique because, if the allograft commissural posts are aligned with the nonequidistant host commissures, leaflet prolapse and poor coaption would occur and the incidence of valve incompetence would be unacceptable. This can be minimized if the noncoronary sinus aortic wall of the graft is retained. The distal suture line is horizontally placed around the host sinotubular ridge as advocated by Ross and associates [12] for the implantation of all allografts (Fig 5).

The allograft may still be inverted for the horizontal first-suture layer but is not usually rotated to leave the noncoronary sinus wall opposing the host noncoronary sinus. Rotation can still be done but the coronary orifice of the graft would need to be ligated or sutured.

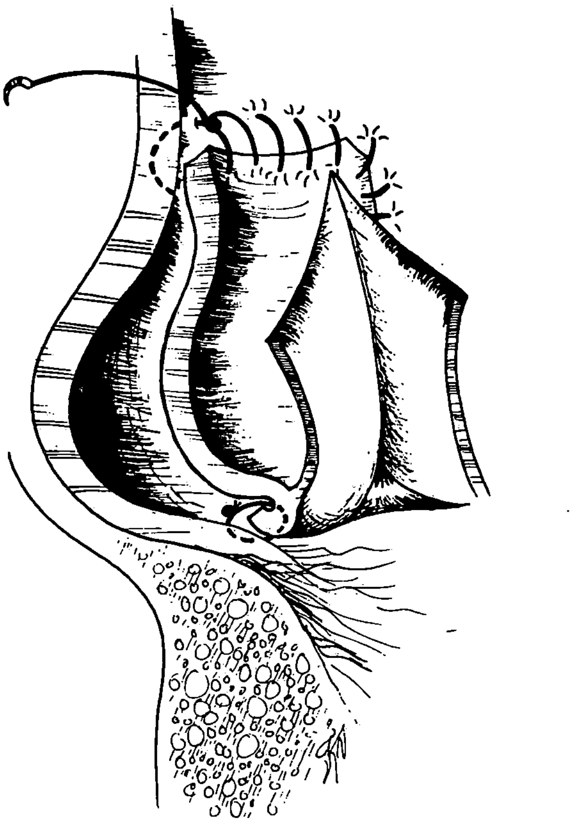

Fig 5. Retention of the allograft noncoronary sinus wall for implantation in the aneurysmal noncoronary aortic sinus.

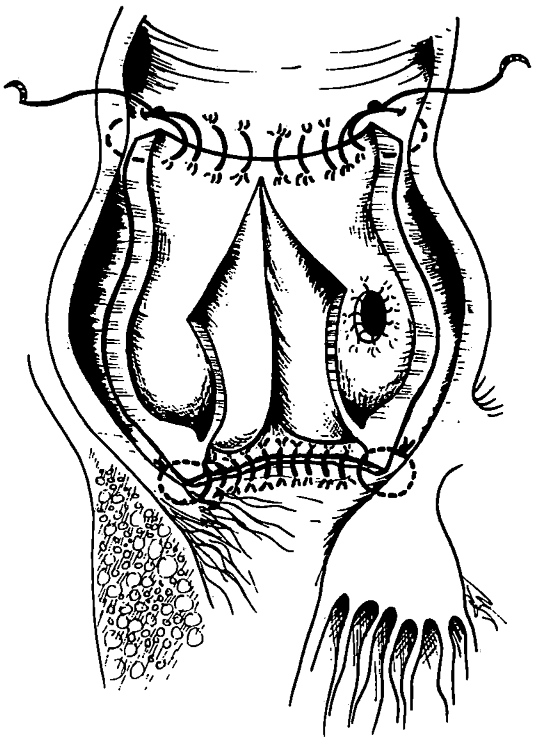

Fig 6. Moderately large aortic annulus: allograft inclusion miniroot technique.

The excess aneurysmal sinus wall can be excised, but it is usually incorporated into the generous aortotomy closure which is begun before the allograft sinus wall is sutured distally into place. If the space between the two walls is excessive, it is probably wise to insert one or two through-and-through mattress sutures to minimize this space.

Retaining the allograft sinus wall reduces the chance of malalignment of the three valve pillars and provides a competent valve.

Moderately Large Aortic Annulus (Valve Annulus Diameter Greater Than 30 mm)

An annulus of this dimension is seen in the patient with symmetrical trileaflet rheumatic valve incompetence in whom there is leaflet retraction and annular dilatation. Some men with calcific congenital valve stenosis have an annulus of 30 mm or more, and poststenotic dilatation is often present. An allograft aortic valve implanted into an orifice of this size often becomes centrally incompetent [2]. Except for those who previously tailored or narrowed the root, most surgeons have discontinued the use of the allograft for the host annulus that is 30 mm or larger.

By using the allograft valve intact with its aortic sinuses as a small cylindrical tube or miniroot (Fig 6), competence can be guaranteed. An allograft with an internal diameter of 24 mm or greater is used for a host annulus of 30 mm.

The allograft is trimmed leaving a longer (4 to 5 mm) cuff below the leaflets, and superiorly the aortic wall is

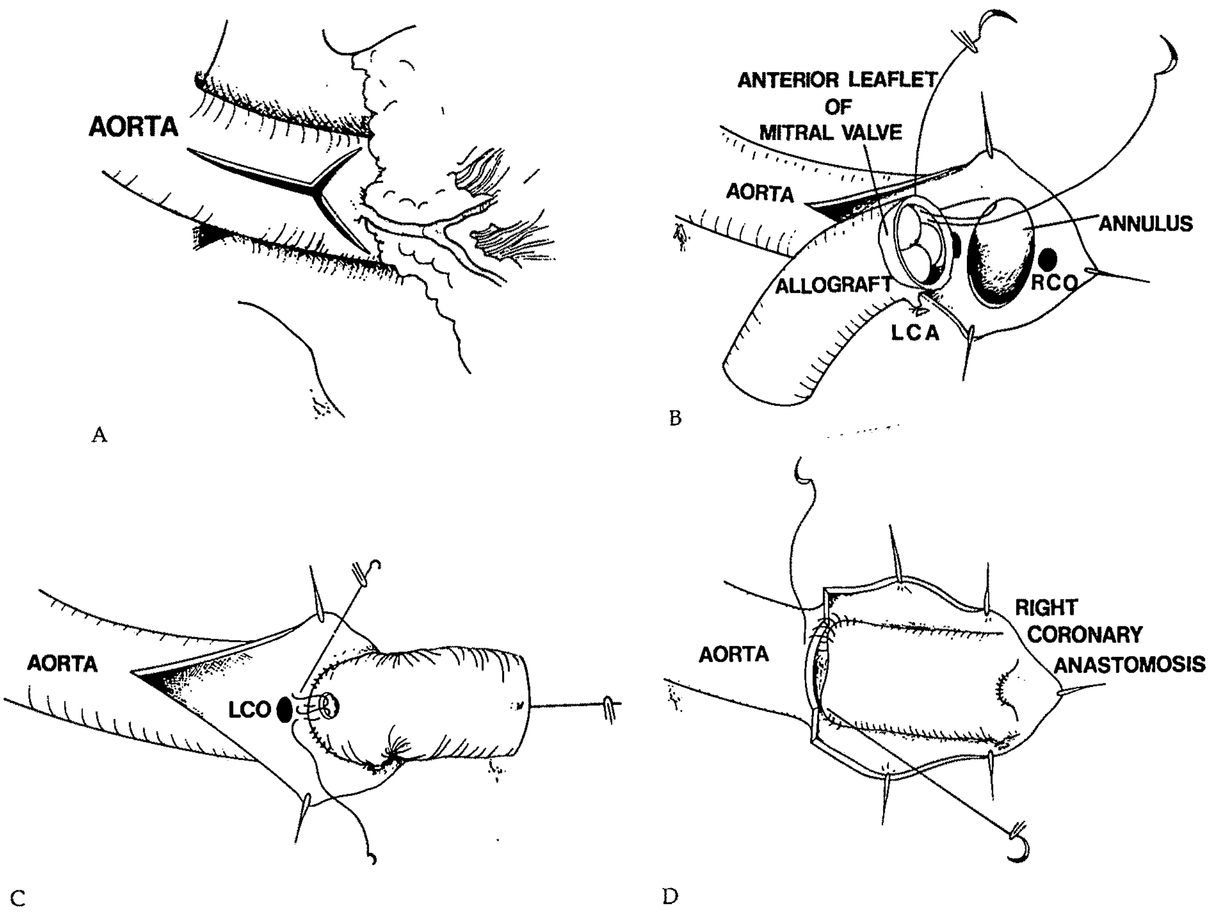

Fig 7. Allograft aortic root replacement. (A) Aortic incision. (B) Proximal anastomosis; left coronary artery of the allograft ligated. (C) Left coronary anastomosis. (D) Right coronary anastomosis; lateral extensions to aortotomy and distal anastomosis. (LCA = left coronary artery; LCO = left coronary ostium; RCO = right coronary ostium.)

transected approximately 5 mm above the top of the commissures. The exposure is generally excellent through an oblique or vertical aortotomy incision. The allograft is rotated 120 degrees and no inversion is performed. The proximal continuous suture line is carried out exactly as for aortic root replacement. The allograft muscle is posterior and a clear view of suturing is obtained. Any trauma to the muscle is minimized but is readily visible should it occur. The sinus wall around the coronary ostia of the graft is excised to create appropriate holes. The coronary anastomoses are performed in the same manner as for aortic root replacement (Fig 7C). Suturing with 4-0 Prolene in a continuous forehand technique taking good close bites of both host and graft tissue is a safe and effective method. Because of the frequent presence of an accessory conal artery arising next to the right coronary artery ostium, this anastomosis circumferentially may have to be large. At this stage, because the allograft is now tethered to the host wall in two sites, it appears distorted.

For the distal suture line, three simple 4-0 Prolene sutures approximate the top of the allograft commissure against the host aorta at the sinotubular ridge. These sutures are tied making allowance for the aortotomy, which can be partly closed first. Almost full-thickness bites of the host aortic wall are taken. Should a suture be full thickness, there is generally no major problem with hemostasis later. To minimize or obliterate the space, it is probably wise to appose the two sinuses in the noncoronary position, either by inserting two mattress sutures as for the large noncoronary sinus technique or by tacking the allograft to the host wall as the aortotomy incision is closed.

This is an inclusion allograft miniroot replacement that provides competence of the aortic valve in a large annulus that was once a contraindication to the use of free-hand allograft implantation.

Large Annulus With or Without Primary Aortic Wall Aneurysm—Allograft Aortic Root Replacement

The indications for aortic root replacement include primary aneurysmal aortic wall disease due to annular ectasia and medionecrosis. The aortic valve leaflets are usually normal although sometimes bicuspid, and the valve is centrally incompetent. Other less common causes of aneurysms are syphilis, arteriosclerosis, healed endocarditis, and rare cases of aortitis (rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, and fragilitas ossium). Left ventricular outflow tract obstruction [14] and aortic root destruction due to active infective endocarditis [18, 19] may be additional indications for allograft root replacement.

At operation [20], as the host aorta is not wrapped around the allograft, the aortic incision includes an additional medial extension toward the left side of the right coronary ostium, giving excellent exposure of the aortic root (Fig 7A).

During the trimming of the allograft, the proximal muscle cuff is left thicker (3 to 4 mm) and longer (4 to 5 mm) than for the previous techniques. The allograft is rotated anticlockwise 120 degrees, which brings the allograft right coronary artery ostium adjacent to that of the host left coronary orifice and the ventricular muscle cuff posteriorly, where more accurate visualization of suturing of this potentially friable tissue can be performed. Accurate alignment of the tissues during circumferential suturing can be facilitated by placing three equidistant simple 5-0 black silk marker sutures in both the allograft lower rim and the corresponding points on the host annulus. Suturing with 4-0 Prolene on a large needle begins on the left side of the proximal allograft rim, passing the needle forehand from outside to inside. This position is at the base of the allograft commissure between its right and noncoronary leaflets (Fig 7B). All continuous suturing (2 mm apart) is with forehand technique and, on completion of the posterior segment, the anterior arm of the suture is begun. It is emphasized that the suture bites must be 2 mm apart to ensure hemostasis.

The allograft left coronary artery is ligated or closed with 5-0 Prolene. The right coronary artery of the allograft is excised to create a hole for the anastomosis of the left coronary ostium. Occasionally a new hole in the allograft sinus has to be made. The allograft is folded forward and the anastomosis is performed with 4-0 Prolene on the small needle (Fig 7C). An appropriately positioned hole in the noncoronary sinus of the allograft is made for the right coronary anastomosis, which is performed in a similar manner, passing the suture forehand through the allograft and then through the host aorta to begin the posterior suture line.

The distal aorta anastomosis is facilitated by improving exposure with lateral extensions to the upper end of the aortotomy (Fig 7D). Because the rotated aorta curves to the right, the allograft should be bevelled and sutured with a small amount of tension on the left inner wall to prevent inward kinking on the left-hand side [20]. Once again, the continuous 4-0 Prolene suturing is performed with a forehand technique. If all of the ascending aorta requires replacement, the distal end of the allograft is sutured to an appropriate size and length of Vascutek (Renfrewshire, Scotland) Dacron graft.

In this procedure, as with all allograft implantation techniques, buttressing of sutures with felt strips or pledgets is avoided.

Aortic Root Infection

The instantaneous risk of postoperative infection of allograft aortic valves is constant and low, whereas the risk of infection of mechanical and bioprosthetic valves has an initially higher phase of risk that declines to merge with a constant and low risk [1, 21]. It can be inferred from this data that the allograft is the replacement device of choice for the treatment of active aortic valve endocarditis.

If the infection is confined to the native valve leaflets, one of the standard allograft valve implantation techniques can be used depending on the root measurements. However, when annular or subannular abscesses (into mitral valve annulus or into ventricular muscle) are present, the abscess can be closed, left open to the circulation, or excluded from the circulation and aortic root but no infected pocket should be left.

For the extensively destroyed root in which some left ventriculoaortic discontinuity has occurred, Ross and associates [18, 19] have demonstrated the efficacy of total allograft root replacement, excluding abscess cavities from the circulation. In less extensively destroyed roots, it is possible to use the subcoronary implantation technique. Kirklin and colleagues [22] have sutured the proximal and distal sinus of the allograft to the edges of annular cavities, obliterating or roofing over the abscess with allograft tissue. In this limited experience, the allograft has remained competent.

Table 1. Allograft Aortic Valve Replacement: Technical Variations

Host Annulus Diameter	Inversion	Rotation 120 Degrees	Proximal Suture Line
Small (≤21 mm)	No	No	Interrupted
Normal size (22–29 mm)	. . .[a]	. . .	. . .
Asymmetrical, large noncoronary sinus	. . .	. . .	. . .
Moderately large annulus (≥30 mm)	No	Yes	Continuous
Annuloaortic ectasia or ascending aortic aneurysm	No	Yes	Continuous

[a] Where no recommendation is made, the particular variation is unimportant.

Summary of Technical Methods

Table 1 summarizes the essential techniques of whether to invert, rotate, and use continuous or interrupted suture method. Where no recommendation is made, we believe that it is unimportant as to which particular variation is used.

If a continuous suture method is used for the proximal layer, the allograft muscle should not be left anteriorly unless it is inverted to avoid the possibility of damage during suturing. The Ross technique with interrupted sutures avoids this risk. More important factors such as retaining the noncoronary sinus wall of the allograft or matching up the coronary arteries may decide rotation. Even so, an allograft coronary artery or ostium can readily be ligated or sutured and another hole made.

At The Prince Charles Hospital, for the usual subcoronary technique, a continuous suture with inversion and rotation of the allograft is preferred. If both the host and allograft leaflets and sinuses are unequal, the allograft should be oriented to allow the inequality to correspond, by no rotation, 120-degree, or even 240-degree rotation.

For the miniroot inclusion technique, the allograft is rotated but not inverted.

Training of a Surgeon

Implantation of an allograft aortic valve by any of the techniques outlined is straightforward and easily learned. Cardiac surgeons perform many more technically demanding procedures than any of these. However, steps taken to learn these techniques should be disciplined and involve practice on animal hearts. Insertion of a small pig aortic valve into a larger pig heart is an ideal way to familiarize the surgeon with the techniques, the handling of valve tissue, and the art of avoiding leaflet damage.

Conclusion

Long-term durability of the aortic allograft valves, although more influenced by preservation methods, is still partly dependent on precise implantation methods. Consequently, the technical aspects of the procedures must be understood and applied to the appropriate aortic root disease process to result in a competent aortic valve with good leaflet coaption and minimal turbulence.

The surgical techniques to be chosen depend more on the geometrical symmetry than on the etiology of the aortic valve disease. There are five groups of varying annular diameters and symmetry that require modification of technique, namely:

1. Small aortic valve annulus—21 mm or less in diameter
2. The common aortic annulus—22–29 mm in diameter
3. The large asymmetrical noncoronary sinus
4. The moderately large aortic annulus of 30 mm or greater—miniroot inclusion allograft root replacement
5. The large annulus with or without primary aneurysmal aortic wall disease—allograft root replacement.

The available methods of implantation, particularly with the addition of the miniroot inclusion technique, have appreciably widened the use of allograft valves. This valve is now suitable for the surgical treatment of all pathological lesions of the aortic valve and root.

We thank Roberta McVicar and Kerri Rohlf for manuscript preparation, Kerri Nelson for art work, and Jordon Prodanovic for photography.

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CORRESPONDENCE

Another Method for Tunneling Defibrillator Leads

To the Editor:

The recent correspondence by Almassi and Olinger [1] details the use of a Penrose drain to tunnel defibrillator leads during implantation of an automatic implantable cardioverter defibrillator. We describe another method using equipment that normally would be used during a left anterior thoracotomy approach.

A 32F chest tube is tunneled from the pocket to the chest incision. All four lead ends can be placed safely into the beveled end of the chest tube, which is then easily withdrawn down into the pocket. The same chest tube is then used for chest drainage.

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Reference

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Reply

To the Editor:

Using a chest tube is another means for transporting the defibrillator leads into the generator pocket. We happen to like the Penrose drain better because it does not create a large tunnel, which can potentially serve as a route for excess pericardial fluid to get into the generator pocket.

G. Hossein Almassi, MD
Gordon N. Olinger, MD

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Internal Banding for Truncus Arteriosus

To the Editor:

I read with interest the account of the experience of Young and associates [1] with internal banding of the pulmonary artery to palliate truncus arteriosus. I have also had some experience with this technique.

A newborn male infant, weighing 2.9 kg, was seen by our pediatric cardiology group with heart failure. Echocardiogram revealed truncus arteriosus and tricuspid atresia. Our goal was to prepare the patient for an eventual modified Fontan procedure and protect the patient's pulmonary vascular bed from resistance changes without distorting the pulmonary artery anatomy. We therefore palliated the patient using a technique similar to the one described. Using moderate hypothermia and a period of cross-clamp with cold blood cardioplegia we placed a polytetrafluoroethylene patch over the ostia of both pulmonary arteries inside the truncal vessel using 7-0 Prolene sutures. We then punched a 3-mm hole in the center of the Gore-Tex patch using an aortic punch. Postoperative recovery was uneventful, with extubation on the first postoperative day.

The patient was restudied at 15 months of age. At that time he was growing well and asymptomatic. The aortic saturation was 81%. The pulmonary arteries appeared to be good size and were undistorted. The catheter could not be passed through the patch perforation. Pulmonary venous wedge measurements of pulmonary artery pressure ranged from 16 to 20 mm Hg, which is slightly higher than we would have preferred. The child is now 2.5 years of age and remains asymptomatic with good growth and development and is awaiting a modified Fontan procedure.

We continue to believe that truncus arteriosus should be treated with biventricular repair, regardless of age, if the level of pulmonary vascular resistance will permit it. In this case, because of the coexistence of tricuspid atresia, interval palliation was necessary. This may be the ideal indication for the innovative technique described by Young and associates.

Vincent A. Parnell, Jr, MD

Division of Cardiovascular Surgery
Department of Surgery
North Shore University Hospital
300 Community Drive
Manhasset, New York 11030

Reference

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Reply

To the Editor:

My colleagues and I were pleased to see that others have had experience with the internal banding technique described in our article. We were also particularly pleased to see that their patient, as ours, had been afforded excellent palliation with good growth and development of the native pulmonary arteries. We would also agree that truncus arteriosus should be treated with biventricular repair when first seen; however, the internal banding technique described in our case report may afford a level of therapeutic flexibility in complex cases, when it is felt that total repair is unlikely to succeed.

J. Nilas Young, MD

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Percutaneous Venous Cannula

To the Editor:

In a recent case report published in *The Annals* [1] my colleagues and I described a new percutaneous venous cannula. Dr John Laschinger should be credited with the suggestion of utilizing a Malecot tip at the distal end of the venous catheter. I regret the omission and am grateful for his suggestion.

Glenn W. Laub, MD

Deborah Heart and Lung Center
Browns Mills, NJ 08015

Reference

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EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Sixteenth World Congress on Diseases of the Chest and the Fifty-fifth Annual Scientific Assembly, American College of Chest Physicians, Boston, Massachusetts—October 30–November 3, 1989

This meeting is sponsored by the American College of Chest Physicians and the International Academy of Chest Physicians and Surgeons. For information, contact the American College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068; or telephone (312) 698-2200.

Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association, Scottsdale, Arizona—November 9–11, 1989

For information on this meeting, contact Gordon F. Murray, MD, Southern Thoracic Surgical Association, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Sixty-second Scientific Session of the American Heart Association, New Orleans, Louisiana—November 13–16, 1989

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 750-5300.

Twenty-fifth Anniversary—Open Heart Surgery, Jerusalem, Israel—December 28, 1989–January 1, 1990

For information on this meeting, contact Joseph B. Borman, MD, or the Secretariat, PO Box 32390, Jerusalem 91323, Israel; or telephone 972-2-663171 (telex: 26205 TRGBL IL; facsimile: 972-2-665277).

Specialty Review in Thoracic Surgery, Chicago, Illinois—January 8–13, 1990

This course is sponsored by the Cook County Graduate School of Medicine and is accredited for approximately 47 hours in category 1 of the Physician's Recognition Award of the AMA. For information, contact the Registrar's office: within Illinois, 1 (800) 621-4649; outside Illinois, 1 (800) 621-4651.

New Trends in Aortic Valve Surgery (Auto, Allo, Xenografts and Reconstruction), Riyadh, Saudi Arabia—January 17–18, 1990

For information on this workshop, contact Department of Cardiovascular Diseases, King Faisal Specialist Hospital & Research Centre, PO Box 3354, Riyadh 11211, Saudi Arabia (facsimile: 966-1-441-4839).

Esophageal Disorders: Pathophysiology and Treatment, Maui, Hawaii—January 25–30, 1990

This program is sponsored in part by educational grants from Synectics Medical, Inc, and Smith Kline & French Laboratories, and is accredited for 24 hours in category 1 of the Physicians Recognition Award of the AMA. For information on this meeting, contact Department of Surgery, Creighton University, CME Program, 601 North 30th St, Omaha, NE 68131; or telephone (402) 280-4501 (facsimile: (402) 280-4593).

Twenty-seventh Annual Seminar—Coronary Atherosclerosis: New Diagnostic and Therapeutic Approaches, Orlando, Florida—January 28–February 1, 1990.

This seminar is sponsored by the Departments of Radiology and Cardiology, Mount Sinai Medical Center of

Greater Miami, Miami Beach, FL, and is accredited for 20.5 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Lucy R. Kelley, Program Coordinator, Radiology Seminars, Inc, PO Box 143762, Coral Gables, FL 33114-3762; or telephone (305) 674-2810.

Nineteenth Annual Meeting of the German Society for Thoracic, Cardiac, and Vascular Surgery, Bad Nauheim, Federal Republic of Germany—February 22–24, 1990

For information on this meeting, contact Hagen D. Schulte, MD, Professor of Surgery, Department of Thoracic and Cardiovascular Surgery, Heinrich-Heine-University, Moorenstrasse 5, D-4000 Dusseldorf, Federal Republic of Germany.

Twenty-ninth Annual Waring-Durrance Chest Conference, Aspen, Colorado—March 7–10, 1990

This meeting is sponsored by the Colorado Trudeau Society and the American Lung Association of Colorado. For information on this meeting, contact American Lung Association of Colorado, 1600 Race St, Denver, CO 80206.

Thirty-ninth Annual Meeting of the American College of Cardiology, New Orleans, Louisiana—March 18–22, 1990

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone 1 (800) 253-4636.

Aortic Surgery Symposium II, New York, New York—May 4–5, 1990

This symposium is sponsored by Meadox Medicals, Inc, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA and toward the New York Medical Association Certificate in Continuing Medical Education. For information, contact Beverly Farrier, Symposium Coordinator, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660.

Seventieth Annual Meeting of The American Association for Thoracic Surgery, Toronto, Ontario, Canada—May 7–9, 1990

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Eleventh Annual Scientific Session of the North American Society of Pacing and Electrophysiology, San Diego, California—May 31–June 2, 1990

For information on this meeting, contact the North American Society of Pacing and Electrophysiology, 13 Eaton Court, Wellesley Hills, MA 02181; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

The Society for Vascular Surgery/International Society for Cardiovascular Surgery, Los Angeles, California—June 4–6, 1990

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Sixteenth Annual Meeting of The Western Thoracic Surgical Association, Coronado, California—June 20–24, 1990

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the ad. To respond to an anonymous ad, write to Box Number, *The Annals of Thoracic Surgery*, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Cardiothoracic surgeon needed for rapidly expanding program located in a coastal area of the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Respond with curriculum vitae and names of references.

Please respond to Box 102. 102G/C

Cardiothoracic and vascular surgeon, board eligible/certified with experience in adult cardiac thoracic and vascular surgery, wanted to join well established 2-surgeon practice located in South Florida. Send CV.

Please respond to Box 105. 105C/B

Cardiothoracic surgeon to join established group in northwest Indiana (very near Chicago). Excellent salary. Please respond with curriculum vitae.

Please respond to Box 104. 104I/L

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join a large cardiac surgery group in southern California. Must have a minimum of 1 year experience in pediatric and neonate cardiac surgery (including fellowship). Excellent fringe benefits. Respond with curriculum vitae.

Please respond to Box 144. 144E/D

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 172. 172G/K

Cardiovascular and thoracic surgeon, board eligible or certified, to join expanding practice in Rocky Mountain region. Salary leading to full partnership. Please send CV and references.

Please respond to Box 165. 165G/J

Thoracic and cardiovascular surgeon, board certified or eligible, wanted for an established practice in the Midwest with experience in adult cardiac, thoracic, and peripheral vascular surgery. Experience or training in heart/lung transplantation preferred. Initial contact should include curriculum vitae, clinical experience, and names of references.

Please respond to Box 162. 162G/J

Additional cardiothoracic surgeon, BC/BE, to join expanding practice of cardiac/thoracic surgery at 500-bed affiliate of major medical center in Midwest. Skills in internal mammary grafting, valve repair, ventricular assist important, and familiarity with electrophysiological procedures desired. Excellent investigational facilities available with ample opportunity for clinical or basic science research. Private practice with academic affiliation—solid general surgery residency with active medical student participation. Salaried position leading to eventual partnership for the right individual. Send CV.

Please respond to Box 178. 178G/J

Assistant surgeon—Preferably experienced in cardiothoracic surgery to first assist in the operating room. Active adult and pediatric cardiovascular surgery practice in metropolitan DC area. Practice includes arrhythmia and vascular surgery, and cardiac transplantation.

Please respond with curriculum vitae outlining training and recent surgical experience to Virginia Heart Surgery Associates, 3301 Woodburn Road, Suite 301, Annandale, VA 22003.
183H/J

Cardiovascular/thoracic surgeon, BC/BE, needed to join busy expanding private practice in Colorado Springs. Adult and pediatric cardiothoracic and some vascular. Initial contact should include curriculum vitae and references.

Please send CV and three reference letters to E. S. Yee, MD, Director of Cardiovascular Surgery, 1725 E Boulder St, Suite 104, Colorado Springs, CO 80909. 185H/J

Chairperson, Department of Thoracic-Cardiovascular Surgery. Winthrop-University Hospital, a 533-bed tertiary care hospital of SUNY Stony Brook, is seeking candidates to chair its Cardiothoracic Department. Applicants should have strong clinical, teaching, and administrative skills, as well as a commitment to research. The department conducts a fully approved open heart program and performs a large volume of thoracic and vascular surgery. The hospital will support a fellowship program and research activities.

Send CV to Alan Fein, MD, Chairman, TCV Search Committee, Winthrop-University Hospital, c/o 222 Station Plaza No., Suite 400, Mineola, NY 11501. 186H/J

Cardiothoracic surgeon, board certified or eligible, wanted to join established 5-man practice. Experience required in transplantation, adult cardiac surgery, and thoracic surgery. Northwest city with easy lifestyle and outdoor activities. Salary leading to partnership.

Please respond to Box 188. 188H/J

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/A

Three-man group currently performing adult cardiac, thoracic, and vascular surgery in desirable northeast location. Seeking associate leading to partnership. Candidate must be BC/BE.

Please respond to Box 194. 194H/J

Cardiovascular and thoracic surgeon: Position available for a board certified or eligible cardiovascular and thoracic surgeon to join a 3-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association will lead to competitive salary, excellent fringe benefits, and early partnership. Please send curriculum vitae.

Please respond to Box 192. 192H/K

Cardiovascular and thoracic surgeon, BC/BE, wanted for established 3-man group in northeastern Ohio. Adult cardiac, vascular, and thoracic experience needed. Please send curriculum vitae.

Please respond to Box 195. 195I/K

Cardiovascular and thoracic surgeon, BE/BC, to join established private practice in San Francisco Bay area. Practice includes adult cardiac, thoracic, and vascular surgery. Initial contact should include curriculum vitae and references.

Please respond to Box 197. 197I/K

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another physician in private practice in Northeast. Immediate availability. Please respond with curriculum vitae.

Please respond to Box 198. 198I/K

Thoracic-vascular surgeon: Excellent opportunity for a board-certified or board-eligible noncardiac thoracic-vascular surgeon to join group practice on south shore of Long Island. Associated with two large level II 400-bed hospitals, fine recreational facilities, including beaches and boating, quality shools, and excellent homes.

Please respond to Box 148. 148EGIKA

Cardiovascular surgeon, board certified or eligible, needed for busy, private established practice. Primarily open heart surgery with some peripheral vascular and thoracic surgery. Competitive salary and benefits.

Please respond to Box 200. 200IJ

Thoracic/general surgeon, BC/BE, to join board certified thoracic/generalist in a busy noncardiac thoracic and general practice. Present physician is the only thoracic surgeon in this community of 80,000 which has a service population of 250,000. Practice is ideally located just one hour from a major metropolitan area and only 30 minutes from an international airport. First year guarantee, incentive, malpractice, and health benefits provided.

Please respond to Box 201. 201J/L

Clinical Associate in cardiothoracic surgery needed to join the San Francisco Heart Institute. BE/BC required. One or two year position with potential for permanent association.

Send CV to Alex Zapolanski, MD, c/o San Francisco Heart Institute, 1900 Sullivan Ave, Daly City, CA 94015. 215J

The University of Florida is seeking a cardiovascular surgeon to join its major teaching unit in Jacksonville, Florida–University Medical Center. Candidates should be eligible for, or certified by, the American Board of Thoracic Surgery. We are looking for someone with excellent surgical skills and a strong interest in research and teaching. Clinical experience in pediatric cardiac surgery is highly desirable. This is a tenure-accruing position with academic appointment at the Assistant Professor to Associate Professor level depending upon qualifications. Applicant recruiting deadline is November 1, 1989. Anticipated starting date is November 15, 1989.

Applicants are invited to forward curriculum vitae and names and addresses of three references to Eric L. Ceithaml, MD, Chairman, Cardiovascular Search Committee, University Medical Center, 655 West 8th St, Jacksonville, FL 32209. Affirmative action/equal employment opportunity employer. 210JK

Thoracic surgeon, board certified/board eligible, wanted to join board certified thoracic surgeon in a 35-physician private practice multispecialty group in a waterfront community on Tampa Bay. Very active general surgery, vascular, and general thoracic practice.

Please respond to Box 214. 214J/L

Noncardiac thoracic surgeon with vascular surgery experience wanted to join expanding Southern California private practice. ABTS certified or eligible. Terms negotiable. Please send curriculum vitae.

Please respond to Box 203. 203J

Thoracic surgeon. A 220-physician multispecialty prepaid group in Hawaii is seeking a qualified MD to accept the second thoracic surgery position. Must be experienced in adult cardiac, general thoracic, and peripheral vascular surgery. Excellent fringe benefits. Malpractice paid.

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Please respond to Box 205. 205J

Cardiovascular/thoracic surgeon, BC/BE, prefer recent graduate to join 2-man group for private practice, predominantly in cardiac surgery in Orange County, Southern California. Excellent salary. Send CV, with experience and recommendations.

Please respond to Box 207. 207J

Cardiovascular and thoracic surgeon wanted to join busy practice in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308. (Telephone: (305) 561-9781 after 9:00 PM.) 208J/C

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Please respond to Box 209. 209J

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Please respond to Box 213J. 213J

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Please respond to 216, The Annals of Thoracic Surgery, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010. 216JK

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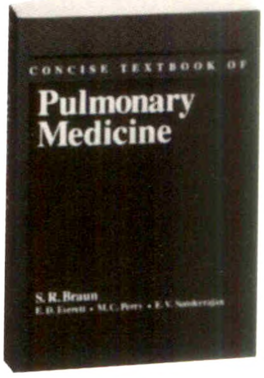

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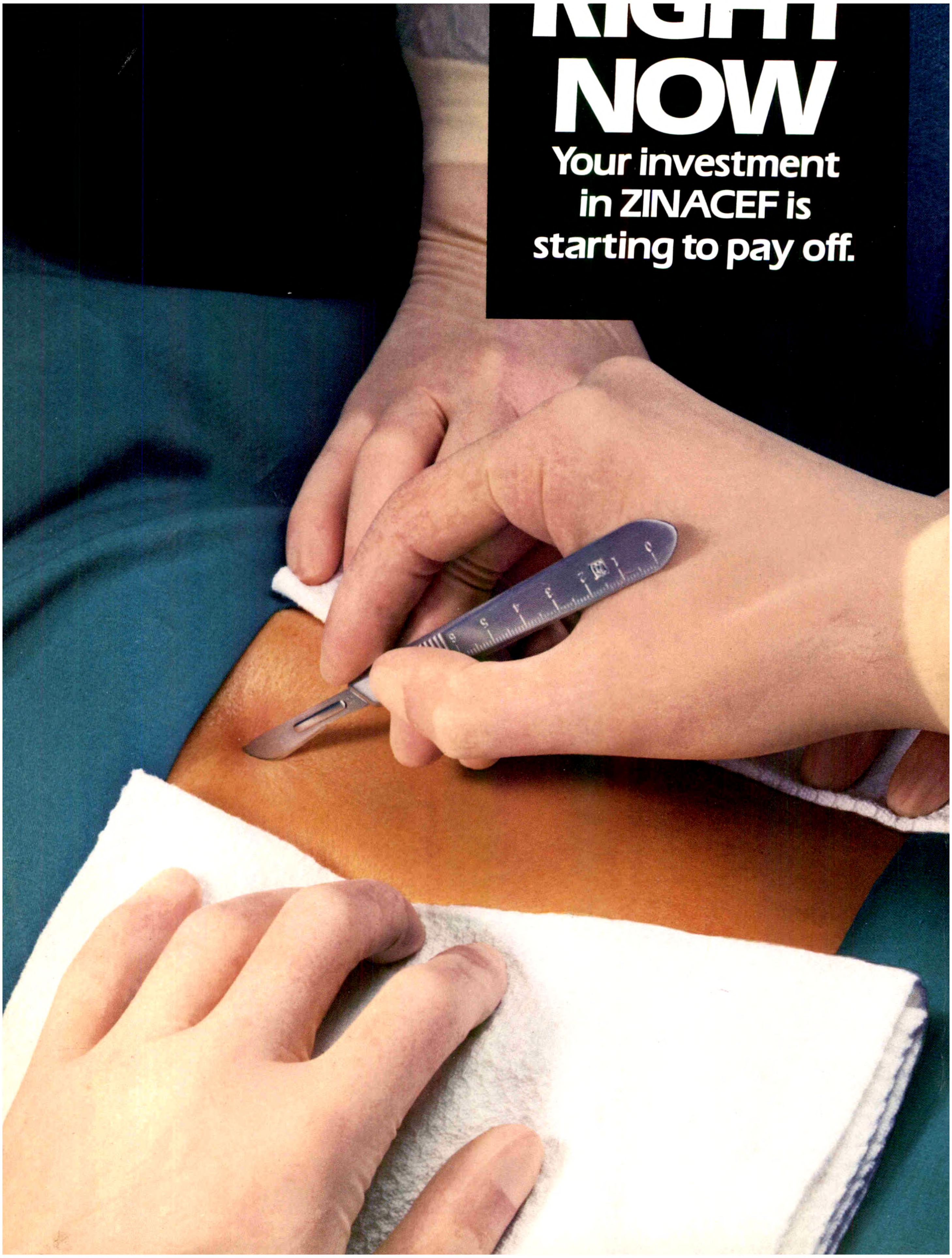
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1. Slama TG, Sklar SJ, Misinski J, et al: Randomized comparison of cefamandole, cefazolin, and cefuroxime prophylaxis in open-heart surgery. *Antimicrob Agents Chemother* 1986;29:744-747.
2. Haley RW, White JW, Culver DH, et al: The financial incentive for hospitals to prevent nosocomial infections under the prospective payment system. *JAMA* 1987;257:1611-1614.

Please consult Brief Summary of Prescribing Information on next page before prescribing or administering ZINACEF.

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sterile cefuroxime sodium

Brief summary. Before prescribing, consult complete Prescribing Information.

INDICATIONS AND USAGE: ZINACEF® is indicated for the treatment of patients with infections caused by susceptible strains of the designated organisms in the following diseases:

1. **Lower Respiratory Tract Infections,** including pneumonia, caused by *S pneumoniae* (formerly *D pneumoniae*), *H influenzae* (including ampicillin-resistant strains), *Klebsiella* sp, *S aureus* (penicillinase- and non-penicillinase-producing), *S pyogenes*, and *E coli*.
2. **Urinary Tract Infections** caused by *E coli* and *Klebsiella* sp.
3. **Skin and Skin Structure Infections** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pyogenes, E coli, Klebsiella* sp, and *Enterobacter* sp.
4. **Septicemia** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pneumoniae, E coli, H influenzae* (including ampicillin-resistant strains), and *Klebsiella* sp.
5. **Meningitis** caused by *S pneumoniae, H influenzae* (including ampicillin-resistant strains), *N meningitidis*, and *S aureus* (penicillinase- and non-penicillinase-producing).
6. **Gonorrhea:** Uncomplicated and disseminated gonococcal infections due to *N gonorrhoeae* (penicillinase- and non-penicillinase-producing strains) in both males and females.
7. **Bone and Joint Infections** caused by *S aureus* (including penicillinase- and non-penicillinase-producing strains).

Clinical microbiological studies in skin and skin structure infections frequently reveal the growth of susceptible strains of both aerobic and anaerobic organisms. ZINACEF has been used successfully in these mixed infections in which several organisms have been isolated. Appropriate cultures and susceptibility studies should be performed to determine the susceptibility of the causative organisms to ZINACEF.

Therapy may be started while awaiting the results of these studies; however, once these results become available, the antibiotic treatment should be adjusted accordingly. In certain cases of confirmed or suspected gram-positive or gram-negative sepsis or in patients with other serious infections in which the causative organism has not been identified, ZINACEF may be used concomitantly with an aminoglycoside (see **PRECAUTIONS**). The recommended doses of both antibiotics may be given depending on the severity of the infection and the patient's condition.

Prevention: The preoperative prophylactic administration of ZINACEF may prevent the growth of susceptible disease-causing bacteria and thereby may reduce the incidence of certain postoperative infections in patients undergoing surgical procedures (eg, vaginal hysterectomy) that are classified as clean-contaminated or potentially contaminated procedures. Effective prophylactic use of antibiotics in surgery depends on the time of administration. ZINACEF® (sterile cefuroxime sodium, Glaxo) should usually be given one-half to one hour before the operation to allow sufficient time to achieve effective antibiotic concentrations in the wound tissues during the procedure. The dose should be repeated intraoperatively if the surgical procedure is lengthy.

Prophylactic administration is usually not required after the surgical procedure ends and should be stopped within 24 hours. In the majority of surgical procedures, continuing prophylactic administration of any antibiotic does not reduce the incidence of subsequent infections but will increase the possibility of adverse reactions and the development of bacterial resistance.

The perioperative use of ZINACEF has also been effective during open heart surgery for surgical patients in whom infections at the operative site would present a serious risk. For these patients it is recommended that ZINACEF therapy be continued for at least 48 hours after the surgical procedure ends. If an infection is present, specimens for culture should be obtained for the identification of the causative organism and appropriate antimicrobial therapy should be instituted.

CONTRAINDICATIONS: ZINACEF® is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

WARNINGS: BEFORE THERAPY WITH ZINACEF® IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE TO DETERMINE WHETHER THE PATIENT HAS HAD PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS, PENICILLINS, OR OTHER DRUGS. THIS PRODUCT SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. ANTIBIOTICS SHOULD BE ADMINISTERED WITH CAUTION TO ANY PATIENT WHO HAS DEMONSTRATED SOME FORM OF ALLERGY, PARTICULARLY TO DRUGS. IF AN ALLERGIC REACTION TO ZINACEF OCCURS, DISCONTINUE THE DRUG. SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES.

Pseudomembranous colitis has been reported with the use of cephalosporins (and other broad-spectrum antibiotics); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with antibiotic use.

Treatment with broad-spectrum antibiotics alters normal flora of the colon and may permit overgrowth of clostridia. Studies indicate a toxin produced by *C difficile* is one primary cause of antibiotic-associated colitis. Cholestyramine and colestipol resins have been shown to bind the toxin in vitro.

Mild cases of colitis may respond to drug discontinuance alone. Moderate to severe cases should be managed with fluid, electrolyte, and protein supplementation as indicated.

When the colitis is not relieved by drug discontinuance or when it is severe, oral vancomycin is the treatment of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should also be considered.

PRECAUTIONS: Although ZINACEF® rarely produces alterations in kidney function, evaluation of renal status during therapy is recommended, especially in seriously ill patients receiving the maximum doses. Cephalosporins should be given with caution to patients receiving concurrent treatment with potent diuretics as these regimens are suspected of adversely affecting renal function.

The total daily dose of ZINACEF should be reduced in patients with transient or persistent renal insufficiency because high and prolonged serum antibiotic concentrations can occur in such individuals from usual doses.

As with other antibiotics, prolonged use of ZINACEF may result in overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Nephrotoxicity has been reported following concomitant administration of aminoglycoside antibiotics and cephalosporins.

Interference with Laboratory Tests: A false-positive reaction for glucose in the urine may occur with copper reduction tests (Benedict's or Fehling's solution or with Clinitest® tablets), but not with enzyme-based tests for glycosuria (eg, Tes-Tape®). As a false-negative result may occur in the ferricyanide test, it is recommended that either the glucose oxidase or hexokinase method be used to determine blood plasma glucose levels in patients receiving ZINACEF.

Cefuroxime does not interfere with the assay of serum and urine creatinine by the alkaline picrate method.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Although no long-term studies in animals have been performed o evaluate carcinogenic potential, no mutagenic potential of cefuroxime was found in standard laboratory tests.

Reproductive studies revealed no impairment of fertility in animals.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in mice and rabbits at doses up to 60 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to cefuroxime. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: Since ZINACEF is excreted in human milk, caution should be exercised when ZINACEF is administered o a nursing woman.

Pediatric Use: Safety and effectiveness in children below 3 months of age have not been established. Accumulation of other members of the cephalosporin class in newborn infants (with resulting prolongation of drug half-life) has been eported.

ADVERSE REACTIONS: ZINACEF® is generally well tolerated. The most common adverse effects have been local eactions following intravenous administration. Other adverse reactions have been encountered only rarely.

Local Reactions: Thrombophlebitis has occurred with intravenous administration in 1 in 60 patients.

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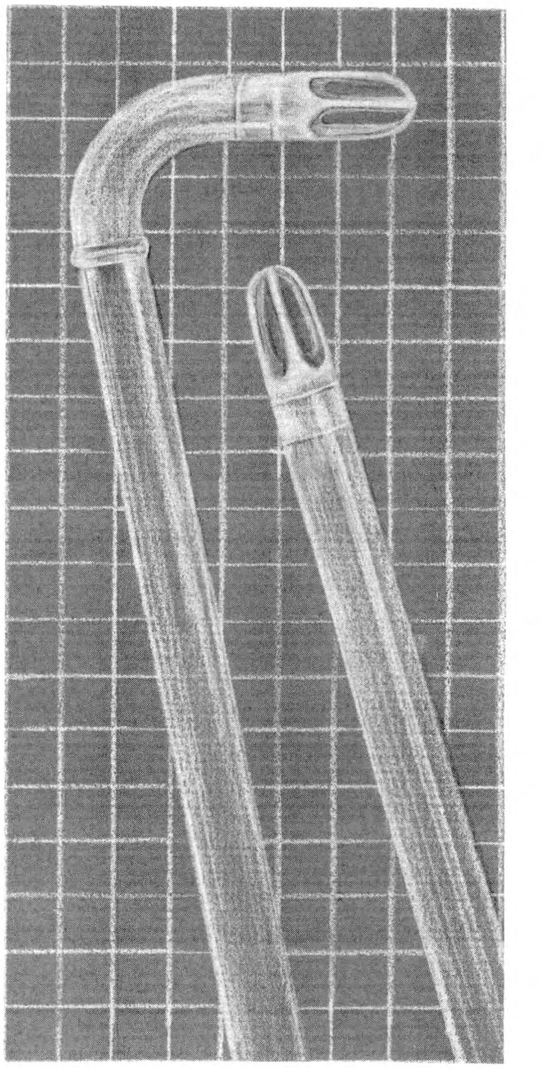

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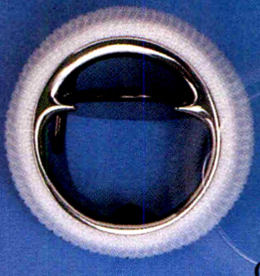

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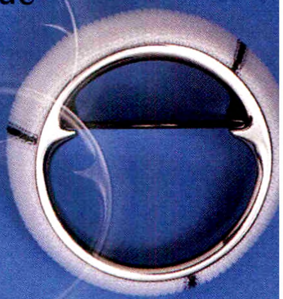

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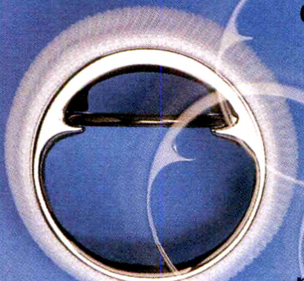

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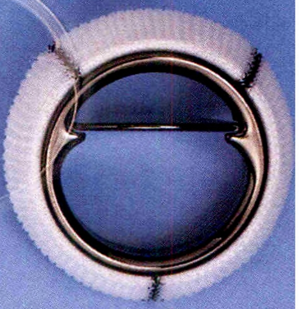

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- ☐ Arrange manuscript as follows: (1) title page, (2) abstract, (3) text, (4) references, (5) tables, and (6) legends. Number pages consecutively, beginning with the title page as page 1 and ending with the legend page.
- ☐ A manuscript for an *original article* ordinarily should not exceed 26 double-spaced typewritten pages, and should be proportionally shorter the more illustrations and tables that are included.
- ☐ The *text* for case reports should be *no more* than 4 double-spaced typewritten pages. If tables or illustrations are included, the text must be reduced. The text for a "how to do it" article should be even briefer, but with detailed illustrative material. Case reports and "how to do it" articles that exceed the allowable length will not be considered for publication.
- ☐ Referencing should be selective and pertain directly to the work being reported. Except in collective and current reviews, comprehensive listings serve no useful purpose and utilize valuable space. As a general guide, original articles should have no more than 20 references; case reports, 8 references; and "how to do it" articles, 5 references.

Title Page

- ☐ Give the paper as short a title as possible. Also submit a short title of 3 or 4 words to be used as a running head. Include only the names of the authors directly affiliated with the work, stating for each the highest academic degree. Include the name and location of no more than two institutional affiliations.
- ☐ If the paper has been or is to be presented at the annual meeting of The Society of Thoracic Surgeons, the Southern Thoracic Surgical Association, or another scientific organization, provide a footnote giving the name, location, and dates of the meeting.
- ☐ At the bottom of the page, type "Address reprint requests to Dr . . ." followed by the last name, exact postal address with zip code, and *telephone number* of the author to whom communications, proofs, and requests for reprints should be sent.

Abstracts

- ☐ Provide an abstract *no longer than 175 words* for an original article. Abstracts for case reports and "how to do it" articles should be shorter (50 to 75 words). An abstract must accompany *every* contribution, except for letters to the editor. Include several (3 to 5) keywords to assist in cross-indexing the article.

Text

- ☐ Generally, the text should be organized as follows: Introduction, Material and Methods, Results, and Comment.
- ☐ *Cite references, illustrations, and tables in numerical order* in the text. (Order of mention in text determines the number given to each.)
- ☐ Spell out, or define in parentheses, all abbreviations, even if they are commonly employed.
- ☐ Give all measurements and weights in *Système International (SI) units.* See "SI Unit Implementation—the Next Step" (JAMA 1988;260:73–6).
- ☐ For statistical nomenclature and data analysis, follow the "Guidelines for Data Reporting and Nomenclature" published in *The Annals of Thoracic Surgery* (1988;46:260–1) and found in the program book for the annual meeting of The Society of Thoracic Surgeons.
- ☐ Type footnotes at the bottom of the manuscript page on which they are cited. Credit suppliers of drugs, equipment, and other material described at length in the paper in footnotes, giving company name and location.
- ☐ Type acknowledgments, including complete grant or subsidy information, at the end of the text before the references.

References

- ☐ Identify references in the text using arabic numerals in parentheses on the line. Do not cite personal communications, manuscripts in preparation, and other unpublished data in the reference list. They may be mentioned in the text in parentheses.
- ☐ Type references double-spaced on a separate sheet. *Number consecutively in the order in which they are mentioned in the text.*
- ☐ Journal references should provide *inclusive* page numbers; book references should cite *specific* page numbers.
- ☐ Double-check references for accuracy, completeness, and nonduplication.

(Continued)

- ☐ Abbreviations of journals should conform to those used in *Index Medicus*. The style and punctuation of the references should follow the formats outlined below:

Journal Article

8. Diehl JT, Eichhorn EJ, Konstam MA, et al. Efficacy of retrograde coronary sinus cardioplegia in patients undergoing myocardial revascularization: a prospective randomized trial. Ann Thorac Surg 1988;45:595–602.

(List *all* authors if 6 or fewer; otherwise list first 3 and add "et al.")

Chapter in Book

12. Katz PO. Achalasia. In: Castell DO, Richter JE, Dalton CB, eds. Esophageal motility testing. New York: Elsevier Science Publishing Company, 1987:107.

Book (Personal Authors or Editors)

18. Larone DH. Medically important fungi: a guide to identification. 2nd ed. New York: Elsevier Science Publishing Company, 1987.

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- ☐ Tables should be typewritten *double-spaced* on separate sheets, each with a table number (arabic) and title above the table and explanatory notes and legends below. Provide a key in alphabetical order to each table to identify all abbreviations used; this key should be placed below any explanatory notes.
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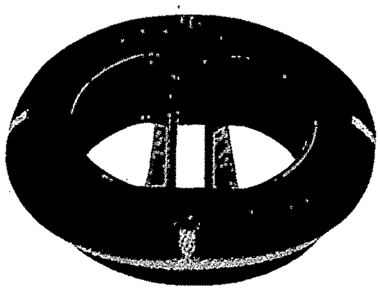

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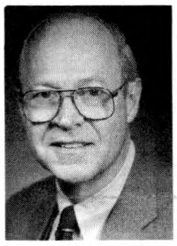
ANNOUNCEMENT

Editorial Board Changes

Four members are retiring from the Editorial Board this year. Nicholas T. Kouchoukos, Martin F. McKneally, and Andrew S. Wechsler have completed nine-year terms, and W. Spencer Payne, a three-year term. The Editor and The Society of Thoracic Surgeons are deeply grateful to these men for their scientific excellence, their dedication, and their many hours of hard work on behalf of *The Annals of Thoracic Surgery*. They were given certificates of appreciation at the Annual Meeting.

Nicholas T.
Kouchoukos

Martin F.
McKneally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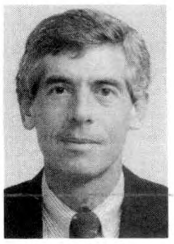
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Wechsler

The Annals is pleased to announce the addition of five new members to the Editorial Board:

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Timothy J. Gardner, Professor of Surgery, The Johns Hopkins University School of Medicine, Baltimore, MD

William A. Gay, Jr, Professor and Chairman, Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT

Mark B. Orringer, Professor and Head, Section of Thoracic Surgery, University of Michigan Medical Center, Ann Arbor, MI

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Timothy J.
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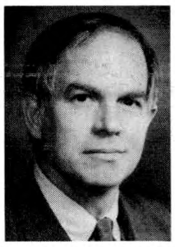
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Pediatric Heart Transplantation

Leonard L. Bailey, MD

Department of Surgery, Loma Linda University School of Medicine, Loma Linda, California

In this issue, Trento and colleagues [1] report the experience of the Pittsburgh group with heart transplantation in children. Thirty-two children (mean age, 9 years) have had heart transplantation since 1982. Excluded from this series are neonates and infants. Fourteen of these children remain alive and remarkably well, although actuarial survival at 6.5 years is a dismal 36%. This figure is significantly lower than expected in their much larger adult population of heart transplant recipients. Excessive perioperative mortality and an unexpected high late mortality from graft rejection are the responsible culprits producing the low actuarial survival. These factors also carry vitally important lessons for all of us committed to seeing transplantation succeed in pediatric patients. It was specifically for this purpose that Dr Trento and his distinguished associates produced this candid, if somewhat vexing report. By permitting you and me to review these preliminary data, the Pittsburgh group understands that we can all learn some critical lessons that will enhance survival for youngsters who receive heart transplants. I laud them for this report. It should in no way discourage us in our mandate to help children with end-stage structural or acquired heart disease. On the contrary, by spelling out some of the dilemmas of managing a pediatric heart transplant service, this report should translate into very much improved results in this arena in the future. In fact, recent reports from other centers suggest that actuarial survival in infants and children may parallel survival in adults [2–4].

If objectivity is difficult in adult recipient selection, it is doubly arduous when one selects infants and children for transplantation. But objective is what we must be. The Pittsburgh team performed transplantation in children with complicated pulmonary arterial anatomy, those with elevated pulmonary resistance, and those bridged to transplantation on extracorporeal membrane oxygenation. What this report tells us is that it is much easier to be human or subjective when confronted with frail, dying children and pleading parents. Each one of us wants so desperately to help, yet the wounds may run even deeper for everyone if selection for transplantation derives from less than objective considerations. I believe that pediatric heart transplant recipients should have normal or surgically correctable pulmonary arterial anatomy and low pulmonary vascular resistance, should be free of untreated infection, and should have other organ systems adequate to insure recovery if substantial morality and morbidity are to be avoided. We all know this to be true, but applying it to infants and children in the emotional heat of the battle remains enigmatic.

Late mortality from graft rejection and accelerated coronary atherosclerosis is a troublesome residual issue among heart transplant recipients of any age, with the possible exception of those who receive transplants in very early infancy [5]. Two factors may help improve late survival among pediatric recipients. Perhaps the most important factor is very close medical and psychological surveillance. This can be expensive, time-consuming, and frustrating. Yet, in our experience, it has been absolutely essential, particularly during the first 6 to 12 months after transplantation. Teenagers are prone to poor compliance, but may be successfully managed if surveillance is sufficiently intense. Second, an immunosuppressive protocol that omits chronic use of oral steroids, tailors cyclosporine and azathioprine dosage to the individual patient's minimal requirements, and makes use of bolus intravenous steroid and/or polyclonal or monoclonal antibodies for treatment of documented graft rejection episodes appears to be advantageous [3, 4]. Exciting newer monoclonal antibodies presently under investigation not only promise to be more effective in abolishing acute and chronic graft rejection, but may also enhance a desired state of selective suppression, if not host immune tolerance [6].

The Pittsburgh report is an eloquent reminder to any institution embarking on a program of pediatric heart transplantation of the energy and intensity required to service such a commitment. Far from being discouraging, a review of their data should help them and each of us committed to helping children with incurable and end-stage heart disease obtain much improved early and late results through heart transplantation.

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Saphenous Vein Graft Valves: "The Bad Guys"

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The report in this issue by Molina [1] is a substantial departure from the standard coronary artery bypass graft technique of using reversed saphenous vein for coronary artery bypass operations. This report deserves careful scrutiny by all practicing coronary artery bypass surgeons for the following reasons.

Flow physics clearly show the inadequacies of what we have termed "coronary-graft discrepancy," ie, a 6- to 7-mm distal diameter saphenous vein graft anastomosed to a 1.5-mm coronary artery with that graft having a 2- to 3-mm proximal anastomosis to the aorta. Our 1-year patency rate of 72% with that graft configuration is significantly below our current average 1-year saphenous vein graft patency of 92%. We have abandoned using grafts in that fashion and have addressed that problem by two approaches. A 3- to 4-cm vein patch has been added to enlarge the proximal anastomosis or, on occasion, the vein graft has been used in a nonreversed fashion as described by Molina.

Valves in saphenous vein grafts became suspect as "the bad guys" as early as 1971. At that time we noted our first case of distal embolism from a cast of thrombus liberated from a saphenous valve to cause a fatal left anterior descending myocardial infarction (Fig 1). This prompted a more careful assessment of saphenous vein graft valves on repeat angiography. The result in a surprising number of instances was the finding of filling defects under the sinuses of saphenous vein graft valves on the repeat angiograms. This prompted the development of a valvulotome [2] designed to divide the central portion of each valve cusp, rendering the valves incompetent as described and shown in Figure 3 in Molina's article [1]. We have found that turbulent flow is clearly diminished after valvulotomy. The valve remnant adheres to the vein wall in the reversed saphenous graft. Postoperative angiography in Molina's study of nonreversed saphenous grafts revealed that the valve site could not be identified in 35% of grafts studied. In the 28 remaining grafts only curled remnants of valve without obstruction or thrombus were found.

Turbulent flow through a normally functioning saphenous vein valve has been identified as a source of thrombi and emboli for many years [3]. Whitney and associates [4], in an elaborate study using time-lapse angiography over an 11-year period, noted microscopically that the majority of stenotic vein lesions occur at valve sites. Furthermore, this group proved that the arterialized saphenous vein valve never fully opens more than 50% in systole. They showed that fibrotic valve stenosis is "acquired" by the increased work of opening and closing with each cardiac cycle as opposed to the natural vein valve movement in the leg environment. Karino and associates [5] studied flow patterns of vein valves and postulated that the incomplete opening of the valve was a hydraulic phenomenon caused by turbulence behind the leaflet. This is corroborated clinically by the occasional angiographic observation of a filling defect under a saphenous graft valve leaflet.

Vascular surgeons were first to address the vein valve problem, voicing great concern with both reversed and nonreversed grafts. They became aware of such abnormalities early on because of their use of completion angiography [6–8].

Our use of the valvulotome in every case since 1975 has resulted in the occasional finding of an inherent cone-shaped valve stenosis that is impossible to detect externally. This occurs in approximately one per 350 vein grafts and would have resulted in early graft failure in every case if not recognized. Confirmation that inherent obstruction at valve sites is not recognizable externally has been reported with the recommendation that all saphenous grafts have an appropriate size probe passed through the lumen before implantation [9]. Reoperation coronary artery bypass, the incidence of which has increased substantially over recent years, has allowed cardiac surgeons to examine pathologically a greater number of explanted grafts. Careful analysis has pointed out with increasing frequency that the saphenous vein graft valve is often the site of the most clinically significant graft disease (Fig 2).

Molina's comments on the use of the valvulotome warrant further amplification. Use of the valvulotome, as with any modality, is only as precise as the effort put forth. The valvulotome should be introduced using loop magnification only after the saphenous vein graft is dilated. Each valve is encountered and clipped to its base. The valvulotome is carefully rotated 180 degrees, and the contralateral cusp is clipped in its central portion. The valve site may be marked with a dot from a sterile marking pen. Traction on the vein adventitia appears less traumatic than actual pull with the valvulotome itself. Two sizes of valvulotomes are available, and a prototype valvulotome with a diamond blade inset is undergoing testing. Insertion of the valvulotome into a side branch must be carefully avoided. This important aspect of graft preparation should under no circumstances be delegated to inexperienced personnel. With special care the saphenous vein graft can be atraumatically prepared for use as a reversed or nonreversed graft with safety, as demonstrated by Molina's 3.5-year follow-up. Likewise, our use of the valvulotome for reversed saphenous vein grafts and an occasional nonreversed saphenous vein graft for 14 years has resulted in no clinical or angiographic evidence

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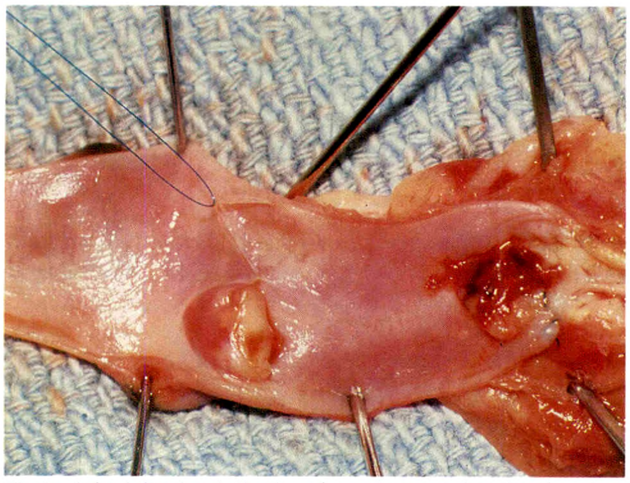

Fig 1. A fatal distal embolization of a saphenous vein valve thrombus into the left anterior descending coronary artery prompted the development of a valvulotome to render the valves incompetent.

of detrimental effect. Molina has pointed out the substantial advantage of automatic air elimination from valvulotomized saphenous vein grafts after the distal anastomoses are performed. The need for introduction of deairing cannulas or needle puncture is eliminated. McCaughan and co-workers [10] reported valvulectomy by venotomy or eversion as the most satisfactory method of addressing the problem. The inherent trauma to the graft, however, makes this technique less appealing.

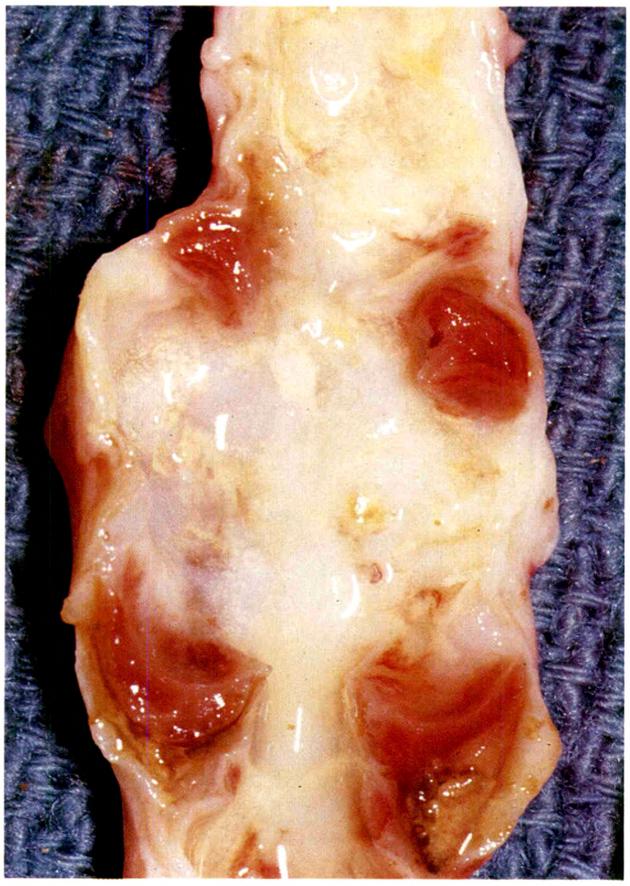

Fig 2. A segment of saphenous vein graft removed 6.5 years after its use for coronary artery bypass. The propensity for graft atherosclerosis to occur at the saphenous vein valve sites is evident.

Most intriguing in Molina's report and a salient fact that requires more corroboration is that a patency of 90% was found after a 19.3-month mean follow-up with restudy of 120 grafts. This is better than the expected 12-month patency rate of standard reversed saphenous vein grafts.

Although there are reports that suggest saphenous vein valves may be beneficial to myocardial perfusion in the early postoperative period, it is apparent from the literature that the long-term ill effects outweigh any early advantages.

In summary, vein valves for vascular surgery procedures and coronary bypass are "the bad guys." Whenever possible, areas of saphenous vein grafts should be used that are free of valves. Segments of saphenous vein graft containing valves near anastomoses should be trimmed off of the graft and discarded whenever appropriate. Otherwise, saphenous vein grafts that include valves should have the valves split and rendered incompetent. Emphasis on proper care with the use of the valvulotome must be accentuated; this point was not addressed in the report by Molina. When nonreversed saphenous grafts are used, it is imperative that both cusps of every valve be divided to their base. Molina has had the courage without completion angiography to pursue the vein valve problem and challenge the concept that coronary vein bypass grafts must be reversed. The goal of a more optimal graft taper may be achieved with this technique. Careful comparison of reversed and nonreversed saphenous grafts is essential in future publications on this subject.

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Aortic Graft-Valve (Composite) Replacement at 20 Years: Wrap or No Wrap? Shunt or No Shunt?

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In 1968, Bentall and DeBono [1] described a technique for replacement of the ascending aorta and aortic valve using an aortic graft-valve (composite) prosthesis. The technique is now widely used to treat a variety of conditions that involve the aortic root. In their early cases, Bentall and associates [1, 2] wrapped the aneurysmal aorta around the graft to promote hemostasis. This maneuver, when used in conjunction with an inclusion technique for the distal aortic and coronary arterial anastomoses, results in a virtually watertight closure. It appears to have reduced the frequency of serious bleeding problems, which were commonly encountered with other techniques that employed separate aortic valve and ascending aortic replacement.

Although wrapping the aorta securely around the graft may reduce postoperative blood loss, it may also mask important sites of bleeding that can result in late complications. In this issue, McFalls and co-workers [3] describe a large pseudoaneurysm of the ascending aortic wall that produced a superior vena caval syndrome 7 years after composite graft replacement of the ascending aorta and aortic valve using the inclusion-wrap technique. It resulted from separation of the graft from the aortic tissue surrounding the left coronary ostium. Although the precise time that this separation occurred cannot be determined, it is not unreasonable to assume that the separation was present at the completion of the initial operation and that persistent leakage of blood into the space between the graft and the overlying aorta produced the large pseudoaneurysm. The long interval before the development of symptoms is not unusual. In our experience [4], the mean time to reoperation of 7 patients with pseudoaneurysms was 41 months, and reoperation was performed as late as 69 months. Because angiography is not performed on all patients after root replacement using the inclusion-wrap technique, the true frequency of pseudoaneurysm formation is unknown. It has been reported to occur in from 7% to 25% of patients [4–8].

Several important modifications of the original procedure of Bentall and DeBono have been introduced that appear to obviate the need for the inclusion-wrap technique. These include preclotting of the composite graft with blood, plasma, or albumin, which eliminates interstitial bleeding and permits more precise localization of sites of bleeding at suture lines [4], interposition of venous or synthetic grafts between the coronary ostia and the tube graft, which reduces tension on the coronary arterial suture lines and permits better access to the annular suture line [9–12], and use of fibrin "glue," which seals the interstices of the graft and bleeding points at suture lines [13]. Use of an "open" anastomotic technique, in which the aortic wall is resected or is not wrapped around the graft until protamine has been administered and control of discrete bleeding sites has been accomplished, is associated with a lower incidence of pseudoaneurysm formation at the coronary and aortic suture lines and of reoperations on the ascending aorta [4, 14, 15].

If the inclusion-wrap technique is used, I believe angiography should be performed in all patients before discharge from the hospital. If a communication between the true lumen and the space between the graft and the overlying aorta is present, it should be promptly repaired. Follow-up examination at 6- to 12-month intervals with chest radiographs and computed tomographic scanning or magnetic resonance imaging of the chest is recommended. Pseudoaneurysms that are detected late after operation should be repaired to prevent catastrophic complications such as the one reported by McFalls and co-workers [3]. Once these aneurysms reach large size, impinge upon the sternum, erode adjacent blood vessels or cardiac chambers, or require emergent operation, operative mortality will be substantial [4, 16].

A modification of the composite graft technique, creation of a fistula between a perigraft space, fashioned from autologous and heterologous pericardium, and the right atrium is described in 2 other case reports in this issue [17, 18]. In both patients, profuse and presumed life-threatening hemorrhage was controlled with this technique with successful early outcomes. The long-term consequences of the resulting left-to-right shunt are not known, as 1 patient died 4 months postoperatively of an unrelated cause and only a 3-month follow-up was available for the other. This technique is a variant of the modification of the original procedure of Bentall and DeBono that was described by Cabrol in 1978 [19]. He wrapped the aneurysmal aorta completely around the prosthetic graft and created a 1.5-cm fistula between the periprosthetic space and the right atrium. In a report of the results with this technique in 100 patients, Cabrol and associates [20] described persistence of the fistula in 2 patients, 1 of whom underwent reoperation to repair a defect in the proximal aortic anastomosis. The true incidence of persistent fistulas and anastomotic leaks was not documented, as routine postoperative angiography or other diagnostic studies were not performed.

Hoover and colleagues [21] applied this technique in 9 of 33 patients having resection of ascending aortic aneu-

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rysms and aortic valve replacement. They created a direct communication or interposed a 6-mm polytetrafluoroethylene graft between the aneurysmal sac and the right atrium or innominate vein. Left-to-right shunts at the right atrial level were demonstrated postoperatively in 5 patients (by oxygen step-up in 4 and Doppler-echocardiographic study in 1) and were considered clinically insignificant. However, congestive heart failure developed in 1 of these patients, and reoperation was required to repair a defect in the aortic suture line. The presence or absence of a persistent shunt was not documented in the remaining 4 patients. Hoover and colleagues hypothesized that flow through these shunts may be insignificant and that they may close spontaneously, but data to support this hypothesis are inconclusive.

The follow-up data of Cabrol and associates [20] suggest that hemodynamically significant shunting is an infrequent occurrence and that closure is common. However, if occlusion of the shunt occurs in the face of a persisting anastomotic defect, the potential exists for development of a pseudoaneurysm. As noted, pseudoaneurysms may not become clinically apparent until many years after operation and operations to repair them are associated with substantial risk.

Although creation of a periprosthetic space to right atrial fistula may be lifesaving and appears to be indicated in situations such as those described by Muehrcke and associates [17] and Blum and co-workers [18], routine or even frequent application of this technique after replacement of the ascending aorta seems inadvisable until the actual incidence and magnitude of residual shunting as well as the frequency of congestive failure requiring reoperation and of pseudoaneurysm formation after spontaneous closure of these shunts are determined. If a shunt is created, noninvasive studies (color flow Doppler-echocardiography, computed tomographic scanning, magnetic resonance imaging) should be used to document these abnormalities. Wider application of the modifications of the original technique of Bentall and DeBono [4, 9–15] should minimize the need for wrapping the aorta around the graft and for shunting to control bleeding.

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Lessons Learned in Pediatric Heart Transplantation

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Between February 1, 1982, and June 30, 1988, 32 children underwent cardiac transplantation for treatment of congenital heart disease (10) and other cardiomyopathies (22). The 6-year actuarial survival was a disappointing 36% because of a high perioperative mortality (12 of 32, 37.5%) and because of five late deaths due to uncontrollable rejection. The perioperative mortality was a staggering 60% (6 of 10) for the patients with congenital heart disease. Four of the 6 recipients with congenital heart disease died because of acute failure of the donor right ventricle. This included 2 patients who required reconstruction of the pulmonary arteries for stenosis secondary to previous systemic-to-pulmonary shunts and 2 others in whom the pulmonary vascular resistances were underestimated because of undetected recent pulmonary emboli (1) and complicated pulmonary vascular anatomy (1). Five of the six late deaths were due to rejection-related events, and all were patients with acquired cardiomyopathy.

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The Pediatric Heart Transplant Program at the Children's Hospital of Pittsburgh was started in 1982 as a natural extension of the Adult Transplant Program that had already been in progress for the previous 2 years [1]. Pediatric patient selection, perioperative care, and long-term follow-up have been under the direction of the same physicians involved in the adult cardiac transplant program. This has allowed us to take advantage of the substantial experience accumulated over the past 6 years with the much larger number of adults who underwent heart transplantation, and it has also assured a homogenicity of care that makes possible meaningful comparisons of the results of heart transplantation in adult and pediatric patients.

Results obtained in our 32 pediatric patients, as compared with the 363 adults who underwent transplantation in Pittsburgh during the same period of time, have been similar in terms of life expectancy of the patients who survived the procedure, side effects of immunosuppressive therapy, and quality of life [2]. However, the children have suffered a high perioperative mortality and a disproportionate incidence of late death owing to rejection or rejection-related events.

Patient Population and Recipient Selection

Criteria for pediatric patient selection for transplantation have been similar to those followed for adult recipients [3] and include end-stage heart disease with a life expectancy of less than 6 months.

Between February 1, 1982, and June 30, 1988, 32 children aged 3 months to 17 years (average age, 9 years) have undergone heart or heart-liver transplantation. Idiopathic cardiomyopathy (15 patients) and palliated congenital heart disease with irreversible myocardial dysfunction (10 patients) are the most frequent causes of end-stage cardiac disease (Table 1). Two patients were treated for acute viral myocarditis. Three others were treated by combined heart-liver transplantation: 2 for primary hypercholesterolemia with ischemic cardiomyopathy and 1 for intrahepatic biliary atresia and dilated cardiomyopathy. A 12-year-old girl required heart transplantation because of end-stage cardiomyopathy caused by adriamycin, and a 12-year-old boy received transplantation because of a primary angiosarcoma of the heart.

Contraindications to heart transplantation in children are the same as in adult patients and have been described previously [4]. As we learned from the adult program, selection of recipients has an obvious impact on survival. Patients with high pulmonary vascular resistance (PVR), previous pulmonary emboli, or complicated pulmonary artery anatomy requiring reconstruction at the time of transplantation have been accepted in our program, but these patients often had a bad outcome, which has significantly affected our overall results.

Survival

The 6-year actuarial survival for the 32 patients who underwent heart transplantation is a disappointing 36%. This is significantly ($p < 0.05$) different from the actuarial survival of the adult population (50%) (Fig 1). Fourteen patients are surviving 4 months to 6 years after transplantation. Three have been alive for more than 4 years, 9 are alive after 3 years, and 2 were transplanted less than 1 year ago.

The difference in survival between the adult and pediatric populations is due to the high perioperative mortality in the latter group (37.5% versus 20%). Children with congenital heart disease had a much higher perioperative mortality (6 of 10, 60%) than those with acquired cardiomyopathy (6 of 22, 27%).

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Table 1. Pediatric Heart Transplantation Between February 1, 1982, and June 30, 1988: Patient Population

Diagnosis	Number	Alive	Dead
Idiopathic cardiomyopathy	16	7	9
Congenital heart disease	10	4	6
Acute viral myocarditis	2	. . .	2
Ischemic cardiomyopathy	2	1	1
Adriamycin toxicity	1	1	. . .
Cardiac angiosarcoma	1	1	. . .
Total	32	14	18

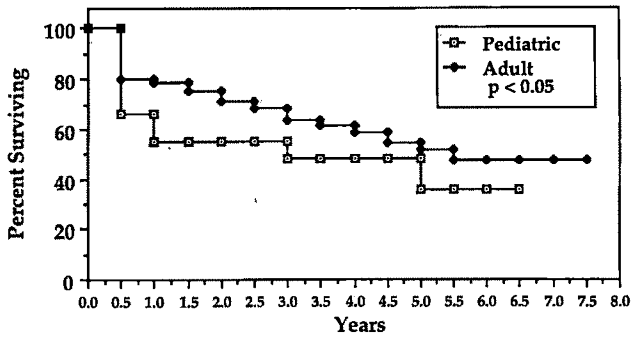

Fig 1. Actuarial survival of pediatric and adult population between February 1982 and June 1988. The difference is statistically significant and is due to the high perioperative mortality of the pediatric population (37.5% versus 20%).

Four of the 6 recipients with congenital heart disease died of acute failure of the donor right ventricle (Table 2). Of these 4 patients, 2 required reconstruction of the pulmonary arteries due to stenosis secondary to previous systemic-to-pulmonary shunts (Pott's shunt and bilateral Blalock-Taussig shunts), and 2 had PVR underestimated because of unrecognized recent pulmonary emboli and complicated pulmonary vascular anatomy, respectively. The other 2 recipients with congenital heart disease also were complicated cases as they required total circulatory support by extracorporeal membrane oxygenation for two and three days, respectively, before transplantation. One of these patients died of biventricular failure of the transplanted heart, and the other died of severe pulmonary hemorrhage (Table 2, patient 6). Extracorporeal membrane oxygenation was instituted in these 2 patients because of inability to wean them from cardiopulmonary bypass at the time of repair of their congenital anomalies (anomalous origin of left coronary artery and ventricular septal defect with anomalous muscle bundle, respectively).

Of the 22 patients with cardiomyopathy who underwent heart transplantation, 6 died in the perioperative period and 6 died after hospital discharge (Table 3). Of the 6 early deaths, 2 were secondary to technical problems related to simultaneous multiple organ transplantation (heart-liver), 2 were due to *Pseudomonas* infections, 1 was due to a brain hemorrhage, and 1 was due to a diffuse lymphoproliferative disorder. Of the 6 patients who died after hospital discharge (9 months to 3 years after transplantation), 1 died of lymphoproliferative disease; the other 5 deaths were related to acute or chronic rejection. Two of these 5 patients had documented extensive coronary atherosclerosis (Figs 2, 3) 2 and 3 years after transplantation, as well as chronic myocardial inflammation and fibrosis, and died suddenly of acute coronary occlusion. The other 3 died of uncontrollable rejection several months after transplantation.

Table 2. Early Death After Pediatric Heart Transplantation in Patients With Congenital Heart Disease

Patient No.	Age	Diagnosis	Time of Death	Cause of Death
1	16 yr	Single ventricle Pott's shunt, left PA stenosis	Third postoperative day	Right heart failure, thrombosis of left PA
2	15 yr	Norwood procedure, bilateral PA stenosis	Intraoperative	Right heart failure, failure of bilateral PA reconstruction
3	17 yr	Double-inlet left ventricle after ventricular septation, cardiomyopathy	Third postoperative day	Right heart failure, recent pulmonary emboli
4	9 yr	Tricuspid atresia, bilateral BT shunts, MVR	Intraoperative	Right heart failure, high PVR
5	3 mo	Anomalous left coronary artery, attempted repair, ECMO support	Intraoperative	Biventricular failure
6	4 yr	VSD and anomalous muscle bundle, attempted repair, ECMO support	Intraoperative	Lung hemorrhage

BT = Blalock-Taussig; ECMO = extracorporeal membrane oxygenation; MVR = mitral valve replacement; PA = pulmonary artery; PVR = pulmonary vascular resistance; VSD = ventricular septal defect.

Table 3. Causes of Death After Pediatric Heart Transplantation in Patients With Acquired Cardiomyopathy

Cause of Death	Early	Late
Rejection	. . .	5
Technical problems	2	. . .
Infections	2	. . .
Lymphoproliferative disease	1	1
Brain hemorrhage	1	. . .
Total	6	6

Immunosuppression and Rejection

The immunosuppressive therapy in our pediatric program, always based on cyclosporine, has undergone the same evolution as in the adult program. In the last 3 years we have used a regimen of triple-drug therapy to reduce the toxic effects of cyclosporine [3]: azathioprine is started preoperatively with a loading dose of 4 mg/kg intravenously, methylprednisolone (20 mg/kg) is administered intraoperatively, and prophylactic antithymocyte globulin (1.5 mg/kg/day) is administered for three to five days postoperatively. Cyclosporine, prednisone, and azathioprine are used for chronic immunosuppression. The maintenance dose of cyclosporine has varied from 4 to 20 mg/kg/day to maintain a whole blood radioimmunoassay cyclosporine level that is greater than 700 mg/mL initially. After 6 months, the cyclosporine dose is decreased to target levels of 500 mg/mL by radioimmunoassay and 150 to 250 mg/mL by high-performance liquid chromatography. Prednisone dose has varied between 0.1 and 0.3 mg/kg/day. The same immunosuppressive regimen is used for our adult transplant patients. In our pediatric patients, the linearized rejection rate was 2.3 episodes per 100 patient-days in the first month; this decreased to 0.3 episodes per 100 patient-days in the sixth month.

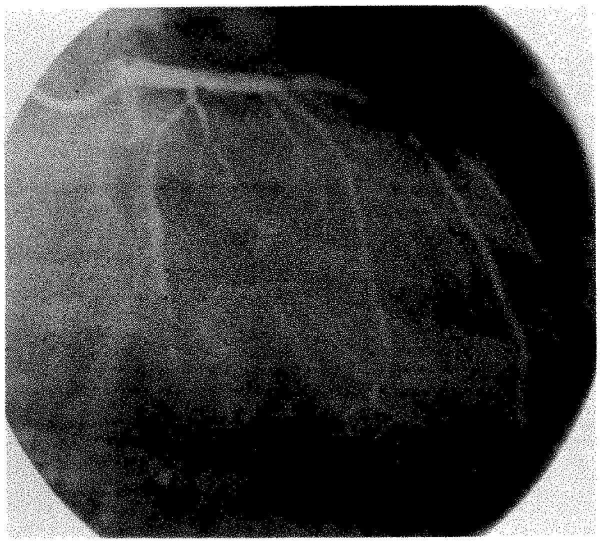

Fig 2. Coronary angiogram shows a tight stenosis of the proximal left anterior descending artery in a 16-year-old boy 3 years after transplantation.

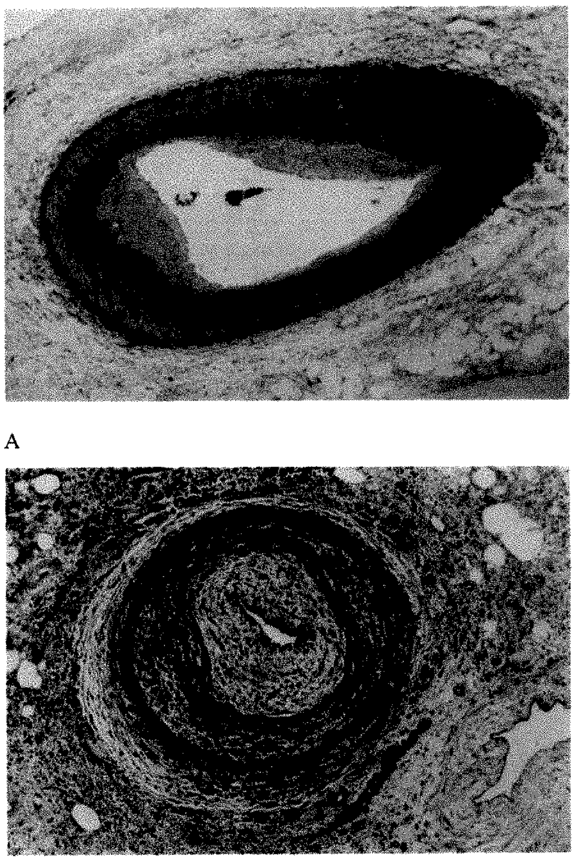

Fig 3. Histological appearance of the circumflex coronary artery (A) and of the left anterior descending coronary artery (B) in the same 16-year-old boy whose angiogram is shown in Figure 2.

Despite the finding that rejection was the principal cause of late death in our children, the incidence of rejection was not different from that of the adult population. Analysis of actuarial rejection rate shows that during the first postoperative year only 25% of the surviving patients were rejection-free versus 31% of the adult patients. The difference does not reach statistical significance. As in the adult population, the frequency of rejection tends to decrease with time, but two episodes of serious and symptomatic rejection occurred 15 months and 2 years after transplantation, both associated with reduction or discontinuation of corticosteroids. Although the reported experience of newborns undergoing heart transplantation indicates that they appear to tolerate an immunosuppressive therapy without steroids well [4, 5], we have not yet been able to withhold steroids from our older transplant patients because of recurrent rejection.

Quality of Life

Follow-up data on the surviving children were reported recently [6]. Growth parameters in children surviving longer than 1 year have shown that linear growth is maintained in most of these patients. In contrast, most patients have shown a marked increase in weight, with only 4 patients on the same weight percentile. Hypertension and abnormalities in renal function related to cyclosporine represent serious long-term concerns. Essentially all surviving children have required antihypertensive therapy, and 8 of the 12 children surviving more than 1 year are currently on a chronic antihypertensive regimen. Combination of diuretics with angiotensin blocking agents have been useful even in the most refractory case. Long-acting calcium channel blockers and α- and β-blockers have also been used with success.

Serum creatinine level usually returns to baseline at about 1 month after transplantation and then increases slowly to reach a plateau at about 1 year. Only 1 patient had a serum creatinine level greater than 1 mg/100 mL, but all the other children had an increase of up to 200% in the serum creatinine level from the baseline level. The abnormalities in serum creatinine level that we have observed are clinically significant, and we are concerned that long-term use of cyclosporine may cause progressive and irreversible renal dysfunction in children.

Despite these side effects of immunosuppression, the surviving children enjoy an excellent physical and emotional rehabilitation, as documented by a follow-up study of 10 of our children who survived transplantation [7]. The study used standardized psychological tests, projective drawings, exercise testing, and parents' interviews to determine the child's daily activities before and after transplantation. In many areas, function improved dramatically after transplantation.

Comment

Our intention in this article was to focus more on the patients who did not survive to determine if the factors responsible for the significant difference between the results of the pediatric and adult programs are specific to our experience and possibly avoidable. An elevated perioperative mortality and a high incidence of late deaths due to rejection have emerged as important features of our review.

The long-term survival for patients who survived operation is very similar in the pediatric and adult population. There is no statistically significant difference in the actuarial survival curves of the two groups after the first 2 months. Improvement in the overall survival of the transplanted children thus can be achieved by decreasing perioperative mortality. Four of the 6 patients with congenital heart disease died of acute right heart failure. Two of these 4 patients had very unfavorable pulmonary artery anatomy and, retrospectively, we would have been wise not to accept them for transplantation. The other 2 patients had undetected high PVR; a more rigorous perioperative work-up might have influenced the decision to select a donor of bigger size or to perform a heterotopic transplantation [8–10]. In reality, the response of the donor right ventricle to increased PVR is unpredictable, and the upper limit of PVR at which orthotopic heart transplantation can still be successful is not known. Criteria developed from the adult transplantation experience suggested that a PVR of more than 6 Wood units or a transpulmonary gradient of more than 15 mm Hg [8, 10, 11] was a contraindication to orthotopic heart transplantation. These criteria are not necessarily valid for the variable body size of children. Therefore, we use a PVR index, which is PVR corrected for body surface area. Recently, the problem of PVR was reviewed by Addonizio and colleagues [10]. In their experience, no mortality occurred if PVR index was greater than 6 Wood units. We successfully performed transplantation in 2 children with PVR index of 9 and 10 U, respectively. Both had reactive pulmonary vasculature, as demonstrated by a decrease in PVR index to 6 during administration of dobutamine. Therefore, a decrease in PVR after administration of pulmonary vasodilators or inotropic agents can be an indication that the patient will be able to accept a heart transplant in orthotopic position, especially if an oversized heart is used.

Heterotopic transplantation has been used only once in our pediatric population, in a 14-year-old boy with idiopathic cardiomyopathy and fixed, elevated PVR. He has survived for 3 years, and follow-up cardiac catheterizations have shown that most of his right-sided output still comes from the native right ventricle and most of the left-sided output comes from the donor left ventricle. On the basis of the good results of heterotopic transplantation in the adult population [11], perhaps we should explore the use of this procedure more frequently even in the younger children. But in contrast to what is reported in the adult patients [11], in whom PVR tends to decrease with time after heterotopic transplantation, in the pediatric population elevated PVR, which does not respond to pulmonary vasodilators preoperatively, may continue to be elevated even after heterotopic transplantation, as was the case in our 14-year-old boy. The transplanted heart will then function mostly as a chronic left ventricular assist device.

The other 2 patients were bridged to the transplant procedure with extracorporeal membrane oxygenation. At present, because of size constraints, extracorporeal membrane oxygenation is the only form of mechanical cardiac support that can be used in the pediatric population [12]. Both of our patients in whom extracorporeal membrane oxygenation was used because of the post-pump left ventricular failure died in the operating room at the time of transplantation because of preexisting pulmonary failure (pulmonary hemorrhage and pulmonary edema) and would not today be candidates for even a heroic transplantation attempt.

The other important aspect emerging from our review is the cause of late deaths, almost all of which were due to rejection. This is significantly different from the experience with our adult population, in which rejection and coronary artery disease were responsible for only 50% of the late deaths; the other 50% being due to infections, malignancy, stroke, liver failure, and other causes. Considering the overall experience between 1982 and 1988, the linearized and actuarial rejection rate of our pediatric population was not different from that of the adult group.

But if we consider only the last 3 years, during which the current triple-drug immunosuppressive protocol has been used, the actuarial rejection rate in the pediatric group has shown that only 36% of the pediatric patients are free of rejection during the first postoperative year versus 47% of the adult population. Two episodes of serious and symptomatic rejection occurred 15 months and 2 years after transplantation, and both were associated with reduction or discontinuation of corticosteroids. For this reason, we have been reluctant to withdraw steroids uniformly from our chronic immunosuppressive therapy.

Accelerated coronary vascular disease in our children remains the major obstacle to long-term graft survival [6]. Prevalence of atherosclerosis in the adult cardiac transplant recipients is estimated to be approximately 20% at 1 year, increasing to 50% at 3 years after transplantation [13, 14]. Data gathered by Pennington and colleagues [15] did not identify coronary atherosclerosis as a major problem in children. This may reflect the short follow-up of most children receiving cardiac transplantation. Coronary atherosclerosis has been a major factor in five of our six late deaths. Two children died of documented acute occlusion of the left anterior descending coronary artery, and 3 died of recurrent or chronic rejection, but autopsy showed that all had severe diffuse coronary artery disease causing ischemic cardiomyopathy. Although pathogenesis of transplant atherosclerosis has not been determined, overwhelming evidence points to an immunological basis [13–16]. We [14] have related the occurrence of two or more major rejection episodes with the development of coronary artery disease. Other factors such as hypertension, lipid metabolism, and obesity may play a substantial role, but a more effective immunosuppressive regimen appears to be the first step in achieving better graft survival.

Lymphoproliferative disease in children receiving cyclosporine has been well described [17]. In our experience, 2 pediatric patients died 2 months and 1 year postoperatively with lymphoproliferative disease involving multiple organs. In both patients, the disease was associated with Epstein-Barr virus infection and with an increased immunosuppressive therapy. The incidence of Epstein-Barr virus–related lymphoma in immunosuppressed children was recently reviewed by Ho and associates [18] at the University of Pittsburgh School of Medicine. It is somewhat higher than in the adult population and is more frequently associated with primary Epstein-Barr virus infection. In our 2 patients, autopsy showed a diffuse lymphocytic infiltration of all organs, including the heart. This diffuse form of the disease appears to be more frequent than the localized form in the pediatric population. The usual treatment of reducing or discontinuing immunosuppression has achieved complete cure in about half of the patients.

Finally, the best aspect of our experience has been the excellent quality of life of the surviving patients. These children have adapted very well to their new situation and are extremely grateful for their new life-style. They can experience and do things that they were never able to do before because of their chronic cardiac illness, which quite often was congenital. But despite these rewarding results, the long-term prognosis of heart transplantation in this age group is, at best, unknown.

In conclusion, even in the pediatric population, heart transplantation is becoming a viable procedure for end-stage congenital or acquired heart diseases. In our experience, the difference between adult and pediatric mortality has been related to acceptance of patients with elevated PVR, previous pulmonary emboli, and severe pulmonary artery anomalies. With proper patient selection and rigorous preoperative evaluation, we can achieve for the pediatric population a perioperative mortality rate approaching that of adult patients. Long-term problems that affect survival, such as hypertension, renal dysfunction, and coronary artery disease, are still unsolved but can probably be addressed by development of new immunosuppressive regimens.

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DISCUSSION

DR LEONARD L. BAILEY (Loma Linda, CA): I thank Dr Trento for sending me a preliminary copy of the manuscript, which I read with considerable interest. Both the manuscript and his presentation have engendered in me a response that is more emotional than intellectual. I'm filled with a combination of astonishment and empathy for my Pittsburgh friends and colleagues who are, I believe, clearly better at what they do than this report implies.

This is, after all, a preliminary report, and its utility has to do with teaching the rest of us where the pitfalls might lie in a pediatric heart transplant program. I believe there must be a better way to effect organ transplantation even in this most difficult of all age groups. I have at least two profound impressions from this presentation.

First, the recipient selection in this preteen and teenage group must be very objective and based on valid, reliable, and current data. Children with more than 5 Wood units of pulmonary arteriolar resistance ought not to have orthotopic heart transplantation in its usual form. I know that Dr Eric Rose at Columbia has been able to see a few children with higher resistances through transplantation. I know also that Dr Magdi Yacomb's group has done the same by using so-called conditioned hearts. But, on the average, results are poor when pulmonary resistance is up. I believe that elevated PVR is a nonnegotiable issue. It is better for every one of us to stay out of that physiological hornet's nest and thereby improve operative survival.

Second, I learned from this report that this age group of recipients on this immunosuppressive protocol may have a higher than expected rate of accelerated coronary atherosclerosis and lethal rejection. There are probably many reasons for this, but I believe that steroids accelerate the condition. Like the Harefield Hospital group, we believe strongly that chronic steroid administration has no place in maintenance immunosuppression of transplanted infants and children. We have been generally successful in achieving this goal, and Dr Yacomb's group never really initiates chronic steroid use in their transplanted children. At last report, Dr Yacomb's group had a 75% overall survival in 36 children operated on since 1984. They have not observed accelerated atherosclerosis, chronic hypertension, and chronic renal failure in anything like the magnitude reported in the present study, and neither have we. I am sure that the recipient compliance in this difficult age group factors into late survival, and these youngsters require the closest of medical and psychological surveillance.

Finally, I wish to add a word about philosophy of management with regard to structural heart disease. If donor supply permits, I believe that babies born with incurable congenital heart disease and normal lungs preferentially should have a heart transplant. The sooner after birth it occurs, it seems to me, the better. Operative survival, quality of life, and durability of therapy may all be enhanced by this approach if our experience at Loma Linda is an indication. Palliative interventions, although frequently necessary, postpone the need for transplantation and thus increase the surgical, immunological, and infectious risks.

I believe we must amplify our interest in infants and children who need heart transplantation. We must learn all we can from candid and honest reports such as the one we have heard today and not be discouraged by these results. Infants and children with end-stage heart disease need our commitment to make heart transplantation not only palliative therapy, but also curative therapy in every sense of the word. We believe we may be edging near to that reality in the newborn and early infant age group. With the help of some of the phenomenal new monoclonal antibodies being studied presently in our laboratory and others, transplantation cures seem inevitable for the older children.

DR CRAIG R. SMITH (New York, NY): I congratulate Dr Trento and associates on another great contribution from their enormous experience. Our experience has been somewhat different at Columbia Presbyterian in New York, where Dr Rose, Dr Reemtsma, and I have performed transplantation in 30 patients between the ages of four days and 18 years.

Although our overall results are quite similar to those of the present study, with a 28% perioperative mortality and a 60% 1-year survival, we differ somewhat in our experience, with 9 patients with congenital heart disease. In this group, there were two perioperative deaths (a 22% mortality), with one death owing to sepsis at 2 weeks and one death in the operating room owing to unequivocal donor failure.

With regard to the discussion in the abstract of what constitutes unfavorable pulmonary anatomy, 4 of our patients had pulmonary arterial anatomy requiring reconstruction at the time of transplantation, accomplished with no perioperative mortality and one late death of rejection. I will briefly describe two examples.

One child with a single ventricle and transposition of the great arteries was treated in infancy with atrial septectomy and pulmonary artery (PA) banding, and underwent transplantation at age 4 years by Dr Rose. At the time of transplantation, the tight band behind the aorta was divided and the arteriotomy was extended into the constricted left PA, with the area patched open with the donor's PA bifurcation. This child is alive and well 4 years later.

Another patient, born with a single ventricle and pulmonary atresia, had a Waterston shunt at age 4 months. When I performed transplantation on this patient at age 17 months, his pulmonary angiogram showed kinking and partial obstruction of the right PA and only trace filling of the left PA. At operation, the Waterston shunt was resected and both PAs were reconstructed with a long pericardial patch. A pulmonary angiogram obtained 3 months later was essentially normal, and this patient is alive and well at 2 years.

Knowing the experience of Dr Trento and his colleagues, when we hear thunder and lightning from Pittsburgh it may be presumptuous to forecast anything but rain. Nonetheless, in this very small subsegment of the transplant population, in which each patient tends to present a unique set of problems, perhaps we should be cautious about making lists of anatomical exclusions. I think patients who are otherwise satisfactory should undergo transplantation if surgical correction has a reasonable chance of achieving acceptable PVR.

My only question for Dr Trento is how do you define unfavorable PA anatomy; isn't it really a matter of achieving an acceptable PVR?

DR TRENTO: I thank Dr Bailey for his kind comments and I compliment the New York group for their excellent results.

It is so difficult to say what is unfavorable PA anatomy. Our 2 patients obviously had difficult PA anatomy. One of them had a previous Norwood procedure followed by bilateral modified Blalock-Taussig shunts, which distorted both PAs at the hilum. Probably at present we would not have considered him a transplant candidate. But I do agree that it is difficult to decide, on the basis of anatomy, whether a patient can undergo an orthotopic heart transplantation.

As far as patient selection, we obviously had a painful experience, and many of those patients that we have transplanted and lost would not now be accepted as candidates. I am referring particularly to the 2 patients who were supported with extracorporeal membrane oxygenation for 3 or 4 days. After this period of extracorporeal membrane oxygenation support, they certainly were not viable candidates for the transplant procedure, because of the severe pulmonary edema and fluid overload.

Nonreversed Saphenous Vein Grafts for Coronary Artery Bypass Grafting

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Between August 1985 and December 1988, valvotomized saphenous vein grafts were used in 365 patients undergoing coronary artery bypass grafting (CABG). In this operation, the femoral end of the vein is attached to the aorta and the pedal end is attached to the coronary artery. Vein diameters measured 8 ± 2 mm at the femoral end, 4.5 ± 1.2 mm at the knee level, and 3.5 ± 1.3 mm at the ankle. Ratios between levels were as follows: knee to femoral end, 0.56, and ankle to femoral end, 0.43. The ratio of knee to femoral end was 0.42 in cases with vein midthigh bifurcation. There were 1,310 grafts implanted (3.6 per patient). In 341 patients, CABG alone was performed, and 24 patients had combined procedures: 11 had CABG with mitral valve replacement, 9 had CABG with aortic valve replacement, 2 had CABG with repair of postinfarct ventricular septal defect, and 2 had CABG with automatic defibrillator implantation. Follow-up (up to 3.5 years) was attained in 97% of patients. For various reasons, 34 patients had a second angiogram between 3 and 41 months postoperatively. Of 120 vein grafts, 108 (90%) were patent. At autopsy, 11 patients with 45 vein grafts had 43 patent and clean grafts and two thrombosed. Use of nonreversed saphenous vein for coronary bypass is recommended. It assures a large proximal anastomosis, natural vein bifurcations can be used with fewer proximal anastomoses, better vein–coronary artery size matching is obtained, and the patency rate is satisfactory.

(Ann Thorac Surg 1989;48:624–7)

When an anastomosis is performed between saphenous veins and coronary arteries, a mismatch exists: the vein is substantially larger than the recipient vessel. Moreover, if the vein is positioned in a reversed manner (ie, with the pedal end of the vein anastomosed to the ascending aorta, and the femoral end to the coronary artery), the mismatch is even greater. Among the hemodynamic considerations affecting the reversed position is the likelihood of developing stenosis at the proximal suture line between the aorta and the vein, particularly when the vein is small and is stretched or flattened to reach and cover the aortotomy site. At the distal end, more turbulence may occur, causing the blood to flow from a small opening in the aorta to a wider distal end connected to a small vessel. This may encourage thrombosis. Nonreversed saphenous vein has been used to bypass the branched renal artery and the hepatic artery [1–3]; in the lower extremities, it has been used in that position for the in situ saphenous vein bypass procedure that offers better results than the reversed operation [4–6].

In about 5% of patients, the saphenous vein bifurcates in the upper part of the thigh, giving origin to two veins that usually run parallel in the thigh down to the knee level. Using the vein in a nonreversed position allows this naturally occurring bifurcation to bypass more coronary branches with only one proximal anastomosis.

With time, fibrotic changes that occur in the veins usually lead to a thickening of the intima, with reduction of its lumen. If the proximal anastomosis is large enough to compensate for this change, fewer possibilities exist of causing early obstruction of the vein. The only potential risk factor in valvotomizing the saphenous vein graft is the question of intimal damage that may induce thrombosis.

Material and Methods

Between August 1985 and December 1988, 365 patients were operated on at the University of Minnesota Hospitals with valvotomized saphenous vein grafts used in a nonreversed position for coronary bypass. Of this group, 341 underwent coronary artery bypass grafting (CABG) alone. In 24 patients, combined procedures were done: 11 had CABG with mitral valve replacement, 9 had CABG with aortic valve replacement, 2 had CABG with repair of an acute ventricular septal defect caused by infarction or a ruptured ventricle, and 2 had CABG with automatic defibrillator implantation. We operated on 47 patients with an acute infarction and postinfarction angina (14%).

Operative Technique

Measurements were obtained of the vein diameter. The cross-sectional area was calculated at three levels: the femoral end, the knee level, and the ankle (Fig 1). In patients with bifurcation or duplication (Fig 2) of the saphenous vein in the thigh, only two levels were measured, the femoral end and the knee level. We tested the vein for leaks by inserting a blunt, beaded-tipped needle at the femoral end and injecting heparinized blood to distend it (Fig 3). A Mills valvulotome (Fig 4) was inserted

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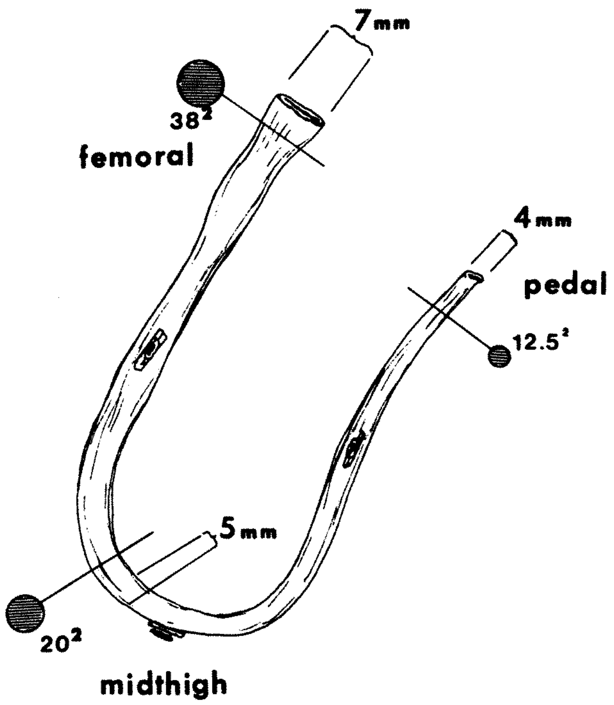
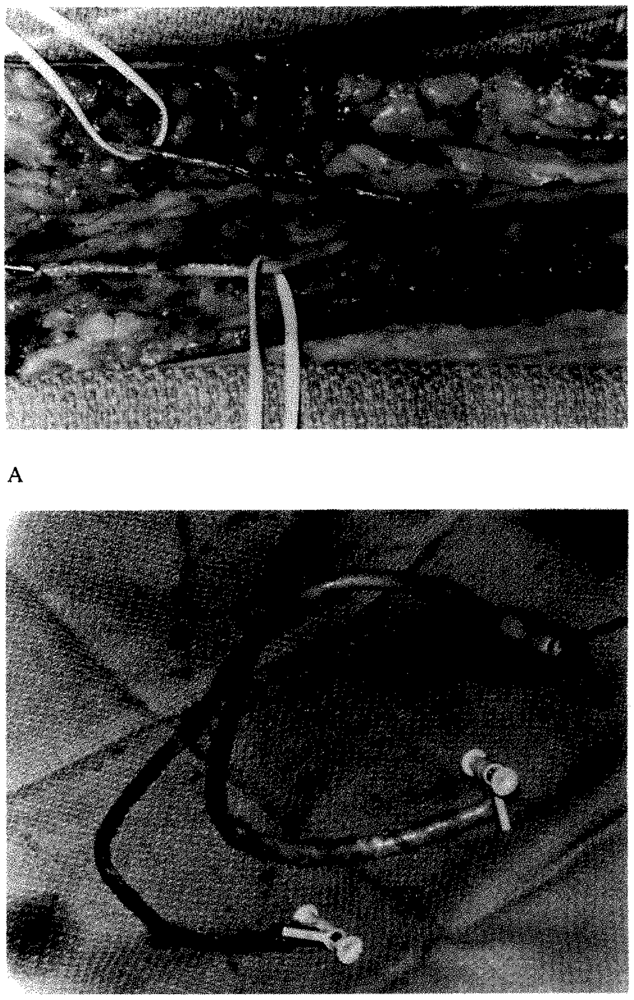


Fig 1. Dimensions found in a routine segment of greater saphenous vein graft with calculated cross-sectional areas (in millimeters).

through the distal end and advanced to the proximal end, severing the valves when it was withdrawn. A bifurcated vein was used every time if the bifurcation was more than 2 cm away from the femoral vein and the branches were long enough to reach the coronary arteries. This situation allowed positioning of the bifurcated graft either in front of or behind the great vessels in the transverse sinus. Whenever possible, sequential graftings were performed with each of the limbs.

In 120 patients, the proximal ends were anastomosed to the aorta first, before the patient was placed on cardiopulmonary bypass. The patients selected for this technique had stable angina, no left main coronary obstruction, and no recent infarction. The proximal saphenous vein anastomosis to the aorta was usually constructed on the right side of the ascending aorta, tailored with a bevel oriented toward the back to pass the saphenous vein in the transverse sinus behind the ascending aorta and with the pulmonary artery exiting over the left atrial appendage. On removal of the side-biting clamp in the aorta, the vein was filled immediately with the blood under systemic pressure, preventing any twisting of the graft itself. When the vein is bifurcated, this maneuver allows optimal positioning of the graft over the vessels to be bypassed and provides measurement of the exact length to the prospective coronary arteries while the heart is still normally distended. The actual flow in the graft can be precisely measured under the patient's normal hemodynamic conditions. In the remaining 245 patients, we connected the distal ends first under cardioplegic arrest and the proximal ends later. With either technique, all the proximal anastomoses to the aorta were performed to a circular orifice made with a disposable punch-instrument (Hancock) 6 mm in diameter, using 6-0 monofilament suture.


A

B

Fig 2. A case of normally occurring bifurcation of the saphenous vein shown in situ (A) and after excision and valvotomy of all the vein valves and being tested by injecting fluid at the femoral end (B).

The sequential anastomoses were performed by making a longitudinal incision in the vein and sewing it cross-fashion with the coronary artery using 7-0 monofilament suture material.

Because the saphenous vein valves are destroyed during preparation of the graft, the air is bled either proximally or distally, without the need to puncture the graft at any level.

Adjuvant Drug Therapy

All patients received Persantine (dipyridamole, 75 mg orally three times a day) and Ascriptin (aspirin plus magnesium hydroxide and dried aluminum hydroxide, 325 mg every day) postoperatively for at least 1 year.

Follow-up

No routine postoperative angiograms were obtained, but 34 patients have had a second angiogram for various reasons in the 3.5 years of postoperative follow-up. Twelve patients (3.2%) were lost to follow-up.

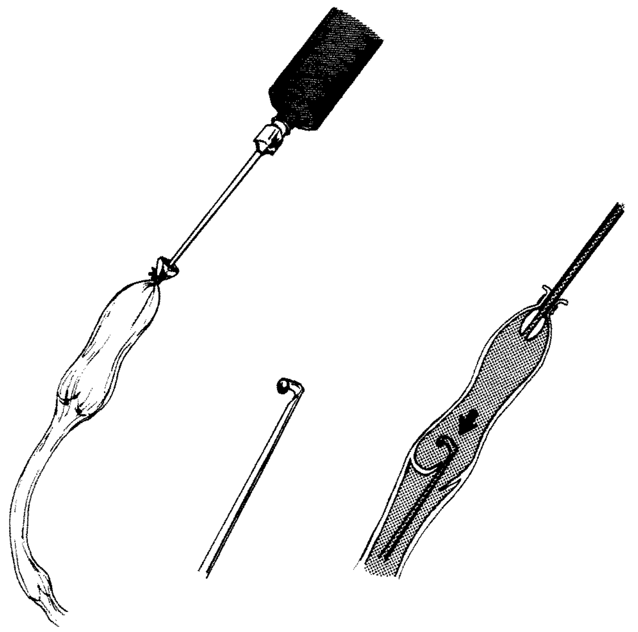


Fig 3. Saphenous vein being injected at its femoral end, showing positioning and technique of performing the valvotomy with a Mills valvulotome.

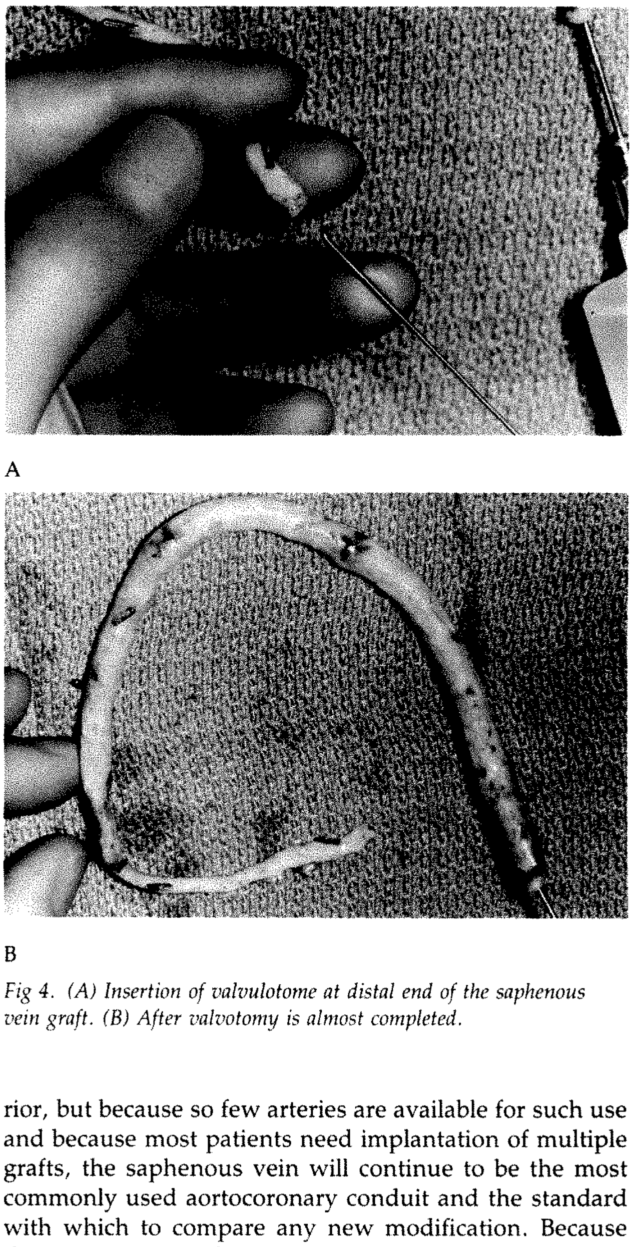


Fig 4. (A) Insertion of valvulotome at distal end of the saphenous vein graft. (B) After valvotomy is almost completed.

Results

Coronary arteriography performed in 34 patients, with 120 grafts, between 3 and 41 months postoperatively showed 90% or 108 grafts to be patent and functioning well. The mean time to angiography was 19.3 months. Angiography showed no evidence of vein valve abnormalities in the grafts. Twelve patients (3.2%) died in the postoperative period, most in the acutely infarcted group (6 of 47, or 12.7%). Their vein grafts, 45 in all, were examined at autopsy: 43 (95%) were found patent without any evidence of thrombosis or stenosis at any level. In 28 specimens, the site of the former vein valve was identified, with small curled remnants of tissue without thrombus or any other pathology. In the remaining 15 specimens, the valve site could not be distinguished from the rest of the vein wall. Only two grafts were thrombosed.

Of the remaining 353 patients, 76 underwent treadmill exercise tests postoperatively; in 2 who had suggestions of ischemia, coronary arteriography was performed. In both, however, the grafts were patent. At various times, ranging from 18 months to 3 years, 7 patients required reoperation due to stensosis in coronary arteries beyond the anastomosis or at the suture line itself or to development of new lesions since the first operation. In 4 of these patients, part of the old saphenous vein graft was removed: in 2 patients at 2.5 years after the original operation and in 2 others more than 3 years after the original operation. The veins showed atherosclerosis and cholesterol deposits in the wall, but determination of the site where the vein valves had been located was impossible.

Comment

Saphenous veins are not the perfect or ideal type of grafts for aortocoronary bypass. Pedicle arterial grafts are superior, but because so few arteries are available for such use and because most patients need implantation of multiple grafts, the saphenous vein will continue to be the most commonly used aortocoronary conduit and the standard with which to compare any new modification. Because the attrition rate of vein occlusion increases with time, many patients will eventually require reoperation when their saphenous vein grafts become nonfunctional. Although atherosclerosis is most commonly blamed for late occlusion of saphenous vein grafts, fibrosis and destruction of the normal morphology of the saphenous vein wall also causes occlusion. Such degeneration may be inherent to removing the vein from its natural position and devascularizing and implanting it in a new position. The vein wall dies either before a new vasa vasorum system can develop or because the diffusion process of cellular nutrition is not sufficient.

Since its inception in the late 1960s, the aortocoronary bypass grafting operation has used the saphenous vein in a reverse position. This was probably an acceptable technique, previously used for femoral-popliteal bypass grafting, because the vein valves could not be destroyed unless the vein was everted totally and the valves were removed with scissors. There was, however, considerable risk of intimal damage, and the maneuver was tedious and time-consuming. Since the design of valvulotomes of the Mills type, this maneuver can be done very easily with minimal or no trauma and within a few seconds [1, 6].

Chin and colleagues [7] showed that vein valves, even in the reversed position, reduce the effective orifice and flow in small veins. When the veins were tested under pulsatile flow, the flow increased only if the vein valves had been destroyed. The smaller the vein diameter, the more significant these changes were. Although the changes observed were significant when the vein was smaller than 2.5 mm in diameter, they may occur in veins somewhat larger but are not detectable by the method of analysis used. It appears that a valvotomized vein, even in the reversed position, has some advantages over its nonvalvotomized counterpart. There is no information in the literature as to whether valves in the reversed position play any role when they are used in aortocoronary bypass. During systole when the left ventricle contracts forcefully, the coronary flow stops within the myocardium and the blood remaining in the epicardial portion of the coronary artery may reflux freely back into the aorta. In this manner, a "washing mechanism" may help to keep a graft open. Whether the presence of a valve in the vein prevents this movement is unknown, but it might play a role in long-term patency.

The valvotomized vein used as a bypass graft functions more like a normal coronary artery. The main advantage of using the vein in a nonreversed position is that the proximal orifice in the aorta can be made larger, thereby preventing fibrosis leading to obstruction at the aortic anastomosis. Second, the vein tapers gradually to a smaller size that connects to the coronary artery. Therefore, the mismatch between vein and artery is diminished and rheology may be improved [8].

In our study's long-term follow-up, there was no evidence that saphenous veins used in a nonreversed position occlude earlier or more frequently than those in a reversed position. In fact, the contrary appears to be the case. Therefore, the minimal trauma associated with the valvotomy is probably of no importance. A clear advantage of the method is the use of normally occurring bifurcations in the greater saphenous vein system. A single proximal coronary anastomosis to the aorta may feed two or more branches of saphenous vein, which can be sequentially attached to the coronary arteries, allowing for multiple grafting and shortening the operating time.

We believe the advantages of using saphenous vein grafts in a nonreversed position for coronary artery bypass are as follows: (1) large proximal orifice; (2) use of natural bifurcation; (3) better size-matching of vein and artery; (4) retrograde air venting; (5) less chance of turbulent flow; (6) equal or better patency rate; and (7) shorter operation.

The use of nonreversed saphenous veins is therefore recommended. No disadvantages have been found in 3.5-year follow-up, and the advantages are quite substantial.

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Experimental Aortocoronary Artery Bypass Grafting Using a CO_2 Laser on the Dog: Acute Experiment

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This study was designed to evaluate the feasibility of using laser tissue welding in aortocoronary bypass operation. Simulated aortocoronary bypass operations were performed on 10 dog hearts supported by extracorporeal circulation. Distal anastomoses between internal mammary artery and coronary artery were achieved using the laser technique, which includes four stay sutures of 7-0 polypropylene and tissue welding between the stays by the laser at a power level of 65 mW. Short-term luminal patency was 100% without stenosis by angiography. Microscopy and histology showed that CO_2 laser caused medial change resulting in fusion with preservation of normal intimal morphology. The aortocoronary bypass operation using a CO_2 laser is technically feasible, and this technique may open up a new method for patients with small peripheral coronary artery obstruction and for pediatric patients with certain conditions (eg, Kawasaki disease).

(Ann Thorac Surg 1989;48:628–31)

At present, CO_2 laser welding is used to anastomose small peripheral vessels in the size range of 0.5 to 1.5 mm [1–4]. Most recently, Frazier and co-workers [5] reported the possible use of a CO_2 laser to anastomose vessels up to 3 mm. The technique appears to be superior to conventional suture anastomosis with respect to small vessel patency. A major concern confronting cardiovascular surgeons working on CO_2 laser tissue welding anastomosis is whether this laser will be able to achieve dependable tissue bonding for anastomosing coronary arteries. This technique may bring good patency results to patients with aortocoronary artery (A-C) bypass with small peripheral coronary artery obstruction and to pediatric patients who need an A-C bypass operation for such conditions as Kawasaki disease. However, the weakness of tissue bonding by CO_2 laser has apparently impeded progress in this area. Therefore, a surgical laser technique has to be established to overcome this problem. In our laboratory, 120 end-to-end and 40 end-to-side laser vascular anastomoses were performed on rabbit carotid arteries, which are similar in size to human coronary arteries, and were followed for up to 1 year with evaluation of patency, aneurysmal formation, and tissue bonding strength related to wound healing [6]. On the basis of satisfactory results with short-term and long-term studies, we initiated A-C bypass studies using the CO_2 laser in the dog and have shown the laser technique to be dependable, at least in short-term experiments. In this paper we report the technical details of the internal mammary artery (IMA) to coronary artery bypass operation using CO_2 laser welding.

Material and Methods

Ten mongrel dogs, weighing 8 to 15 kg, were used for the experiment. All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

All animals were anesthetized with chlorpromazine HCl (5 mg/kg body wt), intubated, and submitted to controlled ventilation. Through the right fourth intercostal space, the heart was exposed and the right IMA was isolated as proximal as possible. After injection of heparin (0.5 mg/kg body wt), the distal IMA was dissected. The IMA was attached to the right ventricular wall to reduce the tensile stress at the anastomotic site. Then, extracorporeal circulation was established with femoral artery and right atrium cannulations. After the aorta was clamped, cardiac arrest was induced chemically under moderate hypothermia. A surgical microscope (×10) was used during laser anastomotic procedure. A low-powered CO_2 microsurgical laser (model TC 3-C 100; Tohoku Ricoh Co, Sendai, Japan), was used for laser anastomosis. This is a continuous mode laser providing 20 to 90 mW with a spot size of 0.18 mm at 10-cm focal length. The laser beam is transmitted through a reflector type of manipulator consisting of seven joints, and guided to the anastomotic site by helium-neon laser (red-colored light).

A side arteriotomy was created on the right coronary artery just proximal to the right ventricular branch and the same size as the diameter of the IMA. Then, four stay sutures of 7-0 polypropylene were placed at heel first, toe second, and then both sides. The segments between the stays were sealed with the CO_2 laser at a power level of 65 mW. During the laser exposure, gentle traction was applied on the two adjacent stay sutures to produce a

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Some of these results were presented at the Motion Picture Session of the Clinical Meeting of the American College of Surgeons, Chicago, IL, Oct 23–28, 1988.

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straight-line coaptation of the anastomotic tissue. The surface at the anastomotic line was kept dry. The end point of laser exposure was a noticeable tissue change to a light brown color and a shrinkage of the vessel edges. The average laser exposure was 10 seconds per segment with a total of 40 seconds per anastomosis, although this duration depended on the surface conditions such as the moisture and adventitia thickness at the anastomotic site. Once the anastomosis was completed, the aortic clamp was released and the quality of the anastomosis was checked.

After the heart was defibrillated, the flow through the IMA was measured with an electromagnetic flowmeter and a uniplane angiogram was carried out. Five dogs were killed immediately after, and 5 were killed two hours after the experiment to study the accumulation of platelets and fibrin. The coronary artery to IMA anastomotic site was then excised and evaluated light microscopically and histopathologically.

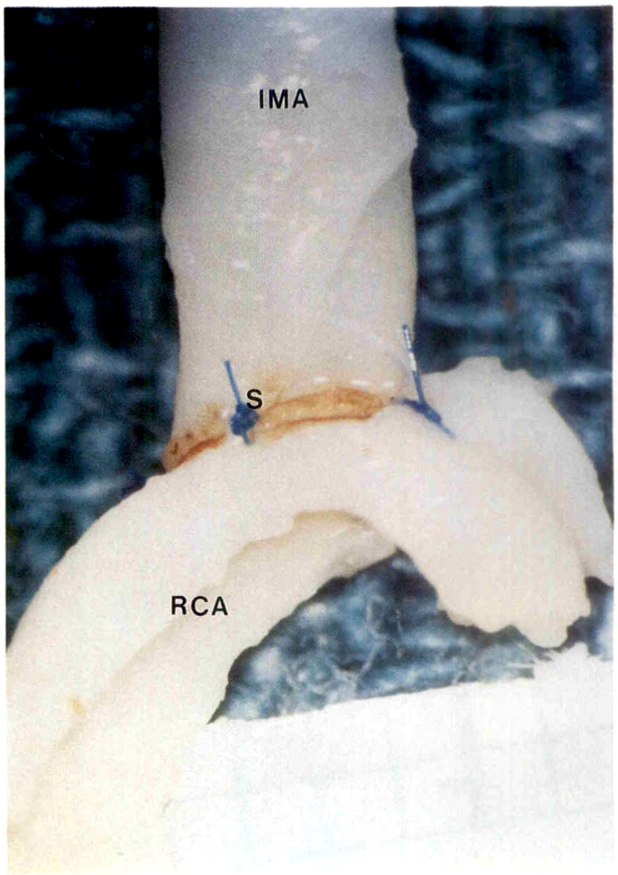

Fig 2. End-to-side anastomosis: four sutures of 7-0 polypropylene are used for stay sutures at heel, toe, and both sides. Carbon dioxide laser energy is delivered between the stay sutures at a power level of 65 mW. The tissue change to light brown color indicates the completion of tissue welding by a CO_2 *laser.* (IMA = *internal mammary artery;* RCA = *right coronary artery;* S = *stay suture.)*

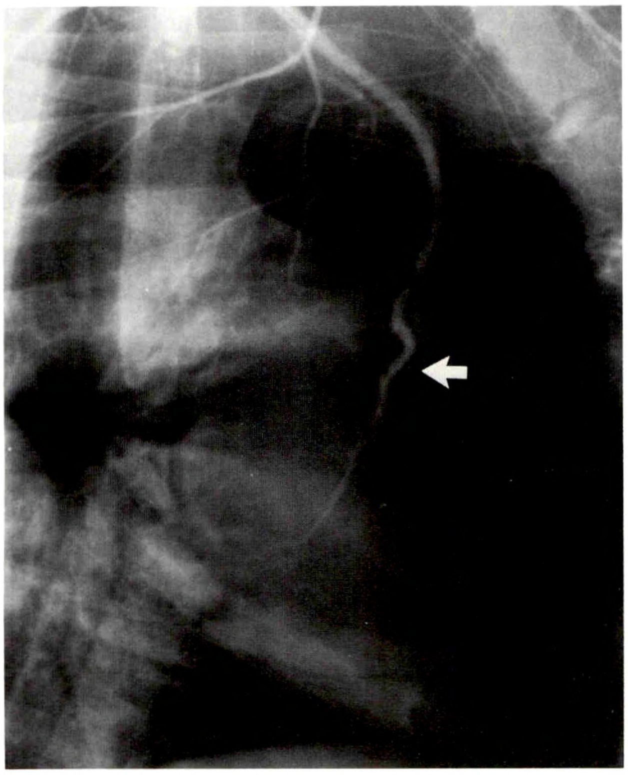

Fig 1. Angiogram of internal mammary to right coronary artery bypass two hours after laser anastomosis. Arrow *indicates the anastomotic site.*

Results

The average aortic cross-clamping time was 20 minutes (range, 16 to 34 minutes), and the mean time used for the laser anastomosis was eight minutes (range, 5 to 13 minutes). In all dogs except 2, the IMA to coronary artery anastomosis was achieved by the described technique. On two occasions, major leakage occurred at the laser-sealed site and had to be controlled by an additional suture. In all cases, a small amount of blood oozing was observed through needle holes, which stopped without intervention. Short-term luminal patency was 100% without stenosis by angiography. An angiogram two hours after the anastomosis is shown in Figure 1. The average blood flow through the IMA was 24 mL/min (range, 20 to 30 mL/min). Light microscopic study from outside of the vessel showed coagulative necrosis and change of tissue to brown-yellowish color on the lased adventitia (Fig 2). The inside view of the vessel showed well-preserved intima, and a small degree of accumulation of platelets and fibrin limited to the stay suture sites. The laser-sealed sites were free of accumulation at two hours after the anastomosis (Fig 3). Histopathological findings showed that adventitia and the shallower layers of the media were fused. In this area collagen fibers were disorganized, which demonstrates the so-called thermal damage by a CO_2 laser, limited to this depth of the vessel wall. The deeper layers of media and intima were well preserved with normal morphology.

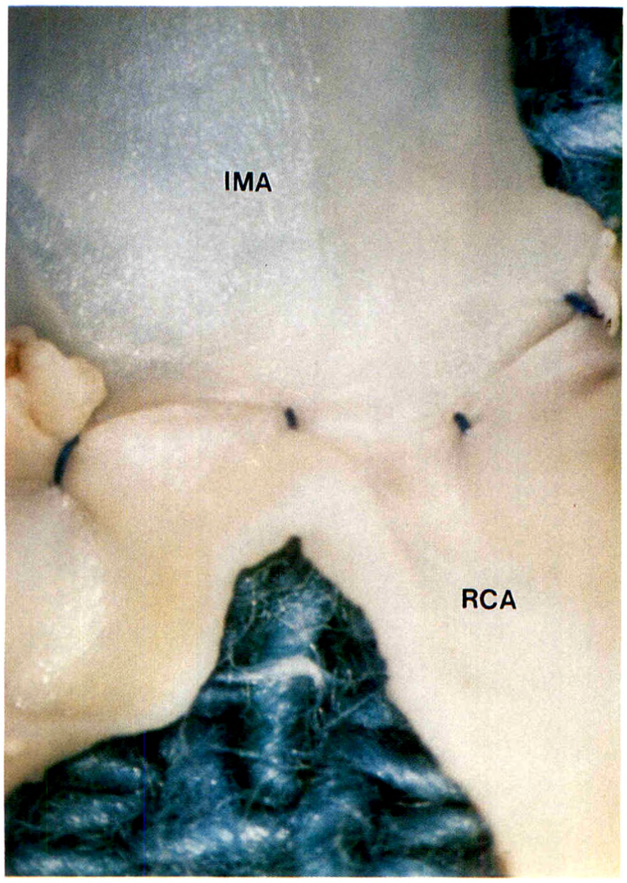

Fig 3. Intimal surface of the laser anastomosis two hours after the anastomosis shows that CO_2 laser anastomosis causes substantially less intimal injury than conventional anastomosis. The anastomotic site is free of accumulation of fibrin or platelets. (IMA = *internal mammary artery;* RCA = *right coronary artery.)*

Comment

The tissue penetration of CO_2 laser is less than 0.2 mm, whereas that of argon laser is approximately 1.0 mm and that of yttrium-aluminum-garnet laser, 4.0 mm. When CO_2 laser energy is delivered to the tissue at the anastomotic site from outside, the tissue welding occurs only in adventitia and media, and the intima is left with normal morphology because of the shallow penetration of the laser. Therefore, considering the full-thickness injury, the CO_2 laser is theoretically most preferable for anastomosing this size of vessels by means of a laser [7]. However, the tissue bonding strength achieved by CO_2 laser is weak, especially in the early period. This strength increases with time after anastomosis, reaching a point equivalent to suture anastomosis in seven days [6].

To increase the tissue bonding strength of the repair, some surgical technical precautions are required. The attachment of the IMA to the right ventricular wall reduces the tensile stress at the IMA–coronary artery anastomotic site. In this situation, it is essential to take a sufficient length of IMA between the attachment point and the anastomotic site. This allows the operator to deliver laser energy easily at the area of the heel stay suture, and also reduces the tensile stress. A length of 7-0 polypropylene, which is easier to manage and stronger than 10-0, is preferable for stay sutures in anastomosing this size of vessels. The stay sutures at the heel and toe were placed slightly further from the vessel edges than standard suture technique, and the stay sutures at both sides were placed slightly nearer to the toe. There were early problems, probably associated with stay sutures, in the previously reported rabbit carotid artery experiment of end-to-side laser vascular anastomosis, but these technical alterations appear to result in improvement in the early disruption rate [6].

A side arteriotomy on the coronary artery should be created as large as the cross-section of the IMA. In performing vascular anastomosis with the laser technique, it is a requirement to appose the vessel edges accurately. If not, fusion will not occur and there may be a risk of aneurysm formation [8, 9]. The surface at the anastomotic site is kept dry as CO_2 laser energy is absorbed nearly 100% by water. Total laser energy should be decided to standardize the laser method. However, the total energy that can reach the tissue completely depends on the surface condition (eg, moisture and adventitia thickness). Optimal magnification has proved to be beneficial as the tissue change caused by laser exposure is the best way to check the completion of the laser anastomosis intraoperatively. Near stay sutures, special attention is necessary to avoid delivering laser energy to the stay suture. Although 7-0 polypropylene is stronger than 10-0 and does not disrupt quickly or easily with a laser shot, if disrupted, the repair is complicated. Because the suture area must be avoided, a very small amount of blood oozing is often observed near the stay sutures even in a successful anastomosis.

Postoperatively, the quality of the anastomosis is checked with the heart fibrillating after the aortic clamp is released. In this experiment, major blood loss from the anastomosis was observed in 2 cases. When major leakage or disruption is observed, additional suture repair is recommended because the nature of collagen has already changed with previous laser bursts and it seems to be hard to reweld the denatured tissue with additional laser bursts. This repair with an additional suture can be performed in the same fashion as in conventional suture anastomosis.

The histological appearance of the laser anastomotic site is almost the same as that on rabbit carotid artery previously reported by us [6] and those reported from other institutions [1, 2, 4, 7]. Minimal intimal injury and minimal accumulation of fibrin by CO_2 laser anastomosis suggests rapid reendothelialization and good patency.

In conclusion, although some technical precautions are required because of the weakness of tissue bonding, A-C bypass operation using a CO_2 laser is technically feasible. The well-preserved intima on microscopic examination supports the excellent patency rates. This technique may provide a new option for A-C bypass patients with small peripheral coronary artery obstruction and for pediatric patients who need A-C bypass operation for such conditions as Kawasaki disease. Further investigation is required to evaluate the long-term results before clinical use.

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Slide Tracheoplasty for Congenital Funnel-Shaped Tracheal Stenosis

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Congenital funnel-shaped trachea is a serious condition, and the survival rate in infants is poor. A slide tracheoplasty is described, with a brief review of other methods of repair. Two cases that demonstrate the operability of congenital funnel-shaped trachea in infancy are reported.

(Ann Thorac Surg 1989;48:632–5)

Congenital funnel-shaped tracheal stenosis is known as an asymptomatic lethal abnormality of early infancy [1]. Few surgical options are available. They include resection with end-to-end anastomosis [2, 3] and various types of tracheoplasties such as esophageal wall patch graft [4], cartilaginous support graft [5], and pericardial patch [6], but serious problems prevent their widespread use.

This article describes a technique devised by one of us (P.G.) that involves transverse division of the trachea at the midpoint of the stricture, spatulating the proximal and distal tracheal ends, and a "slide" tracheoplasty. It appears to be safe and effective, providing an adequate tracheal lumen. The effect of growth on luminal size is not yet known.

Technique of Slide Tracheoplasty

Preoperative tracheoscopic and bronchoscopic examinations are important in defining the site, length, and severity of the stenosis and its relationship to complete tracheal rings. Tracheostomy is avoided.

The technique of slide tracheoplasty is shown in Fig 1. We now prefer to use transverse cervical incision as our approach if the entire trachea is involved. Right thoracotomy may be suitable for localized lesion of the lower trachea, however, especially if the carina is involved or an aberrant pulmonary artery is suspected. The trachea is exposed and is circumferentially mobilized to the carina and divided transversely at the midpoint of its narrowed segment. Ventilation is continued across the operative field into the left main bronchus with a 2-mm or 3-mm Portex endotracheal tube. The distal segment of the tracheal stricture is divided longitudinally along its posterior wall down the length of the trachea as far as the carina if necessary. The proximal end is divided longitudinally along its anterior border, extending across all complete rings to the full length of the stricture. With adequate tracheal mobilization and flexion of the neck, the passage of two traction 5-0 Prolene (Ethicon) sutures at both proximal and distal ends will approximate the two tracheal segments.

A slide tracheoplasty is performed with continuous 5-0 Prolene suture. Ventilation during this procedure continues by an endotracheal tube with intermittent ventilation. We have found a pulse oximeter valuable, but Venturi jet ventilation has proved less safe and efficacious. Ventilation is resumed with the endotracheal tube positioned above the anastomosis. Two sutures are used to fix the chin to the anterior chest wall, keeping the neck in flexion for seven days postoperatively to reduce the tension on the tracheal anastomosis. No laryngeal release procedure is required. The infant is kept intubated during this period to minimize any interference with the repair and risk of major airway obstruction with the neck in flexion.

Case Reports

Patient 1

A 10-month-old male infant, diagnosed as having a small perimembranous ventricular septal defect at age 4 months, sustained a chest infection with increasing respiratory distress and severe stridor. There was no previous history of respiratory trouble. He was intubated and transferred to the Brompton Hospital. Maintaining satisfactory ventilation was difficult, despite a high peak airway pressure of 60 cm H_2O. Bronchoscopy showed funnel-shaped trachea with normal width in its upper half with complete tracheal rings and progressive narrowing in its lower half that would not admit the pediatric bronchoscope. Computed tomography showed no extratracheal abnormality (Fig 2). Limited tracheogram confirmed severe narrowing of the lower trachea extending down to the carina (Fig 3).

Tracheal reconstruction was undertaken through a right thoracotomy, with the stricture ends divided and spatulated to preserve all tracheal tissue and increase the reconstructed lumen. The operation was complicated by an aberrant left pulmonary artery that was acting as a sling around the lower trachea. The tracheal reconstruction was carried posterior to the pulmonary artery to correct the vascular sling. During this procedure, intermittent ventilation/apnea technique was used because only the left lung was ventilated and any surgical manipulation compressed the left pulmonary artery. Immediately after reconstruction, the airway pressure decreased

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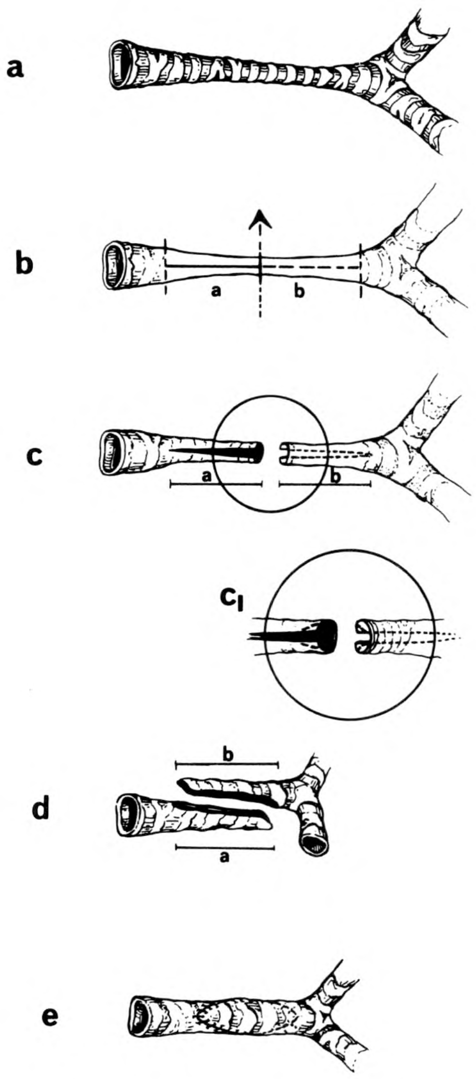

Fig 1. Slide tracheoplasty. (a) Funnel-shaped trachea with complete tracheal rings. (b) Trachea divided at the midpoint of the narrow segment (a = b)*. (c) Both tracheal ends were divided longitudinally and spatulated as shown in (c_1). (d) Lateral view of slide tracheoplasty. (e) Reconstructed trachea with a wider lumen.*

to normal and the lung collapsed readily on expiration (pH, 7.28; carbon dioxide tension, 7.5 kPa; oxygen tension, 28.2 kPa; fraction of inspired oxygen, 0.8).

On the third postoperative day, dense bilateral pulmonary infiltrates developed, eventually confirmed as *Legionella pneumophilia* infection acquired while the infant was an inpatient at the referring hospital. He was treated with the appropriate antibiotics, initially erythromycin and later rifampicin. Despite the high-pressure ventilation needed, the tracheal repair remained intact bronchoscopically 4 weeks after operation. The high inflation pressure required, however, produced pulmonary damage in itself. Despite continuing ventilation and drainage of recurrent pneumothoraces and lung cysts, he died after 5 weeks of intensive care.

Patient 2

A 3-month-old female infant with mild dyspnea and repeated upper respiratory tract infections since birth had had an apneic episode at home. On admission to the hospital, she had cyanotic and bradycardic episodes that worsened when she cried. Her chest radiograph showed right basal collapse. Her ventilatory pattern deteriorated, requiring ventilation. Bronchoscopy at the referring hospital revealed severe tracheal stenosis due to complete small tracheal rings, and the infant was transferred to the Brompton Hospital. Computed tomography showed a progressively narrowing trachea down to the carina, which was slightly widened with early takeoff of the right upper lobe bronchus. Right heart catheterization showed no evidence of pulmonary artery sling.

Preoperative bronchoscopy revealed a 60-mm trachea. Beneath the larynx, the trachea tapered, with severe stricturing starting at 17 mm and continuing for another 35 mm with a diameter of 3 mm. Below this, the trachea opened over the next 8 mm to the carina. The tracheal takeoff of the right upper lobe bronchus was confirmed. Through a transverse cervical incision, the trachea was exposed to the carina, and the stricture was divided transversely at its midpoint. A slide tracheoplasty was undertaken with the technique and ventilation described in the preceding section. The reconstructed tracheal lumen was almost twice the size of its preoperative diameter.

The procedure was uneventful despite the infant's small size. Chin sutures were used to maintain neck flexion for seven days. Chest radiograph showed reexpansion of the right lower lobe. On the eighth postoperative day, she was extubated (oxygen tension, 28 kPa, carbon dioxide tension, 4.7 kPa, fraction of inspired

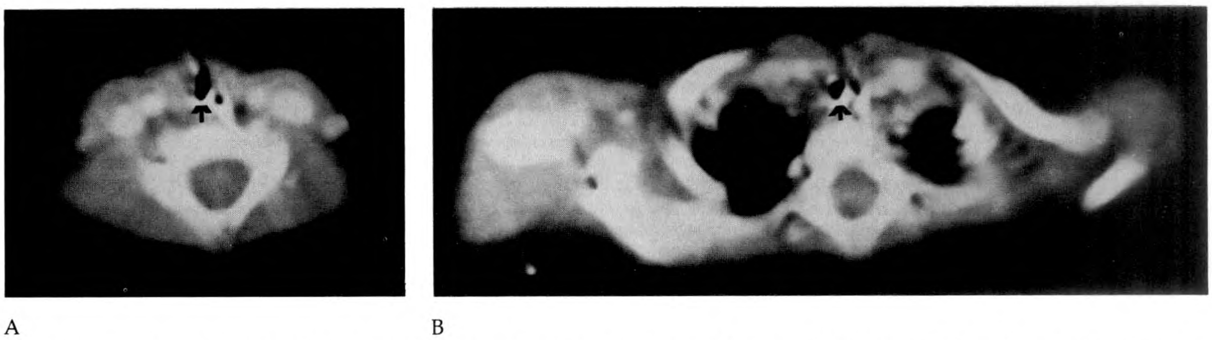

Fig 2. (Patient 1.) Computed tomography of the cross-sectional area of the (A) normal upper trachea (arrow) *(15 mm^2) and (B) narrowed lower trachea* (arrow) *(9 mm^2).*

oxygen, 40%) and required no further respiratory support.

At 12 months follow-up, the baby was thriving and gaining weight. There was no evidence of major airway narrowing, as shown by the normal inspiratory and expiratory time (1.12 and 1.30 seconds, respectively) and the peak inspiratory and expiratory flow (3.2 and 3.3 L/min, respectively). The flow/volume curve was normal. The tracheal tomogram showed an adequate lumen throughout the length of the trachea (Fig 4).

Comment

Congenital funnel-shaped trachea is a life-threatening abnormality [1]. Cantrell and Guild [2] described its pathology in detail. The funnel shape narrows over a variable length but in extreme cases extends down to the carina with absent membranous trachea and complete cartilaginous rings. Its surgical management is similar to that of any long, severe congenital tracheal stenosis. Several surgical options have been described, but survival has been uncommon.

Resection and reanastomosis of long segmental stricture has been described [3]. This requires extensive mobilization to obtain surgical proximity and tension-free anastomosis and is not possible technically in most cases of funnel-shaped trachea, in which more than 50% of the trachea is narrowed [4]. Cantrell and Guild [2] suggested resection of the narrowest part of the stricture at the carina with reanastomosis, but a substantial length of complete cartilaginous rings would still be left intact. Ein and co-workers [5] described a technique involving cardiopulmonary bypass, opening the entire length of the posterior wall of the stenosed trachea and patching the defect with the anterior wall of the esophagus. The infant required long-term tracheotomy intubation to prevent tracheal collapse, however, and subsequently died. Reconstruction of long tracheal stenoses using autologous cartilaginous grafts has been successful [6], but such grafts require long-term endotracheal stent support until fusion occurs between the graft and the tracheal wall. The other complication associated with the graft is compression of major vessels within the mediastinum in small infants. Tracheoplasty with pericardial patch [7] for extensive tracheal stenoses has been used in infants to widen the anterior cartilaginous part. Extracorporeal circulation for respiratory support was advocated for surgical access when the airway was critically narrowed. However, it is our experience that adequate exposure of the trachea can be achieved without cardiopulmonary bypass and its associated risks. The 2-year follow-up has been encouraging, but shrinking of the pericardial patch has been recognized as a potential problem.

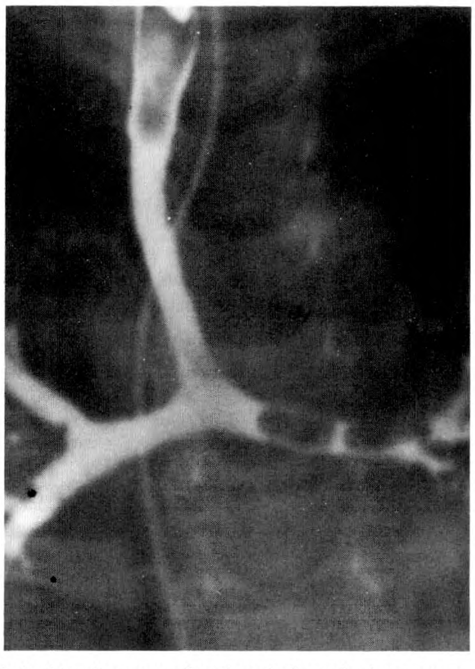

Fig 3. Limited tracheogram of patient 1 showed a long severe narrowing of the lower trachea, which would not admit a pediatric bronchoscope.

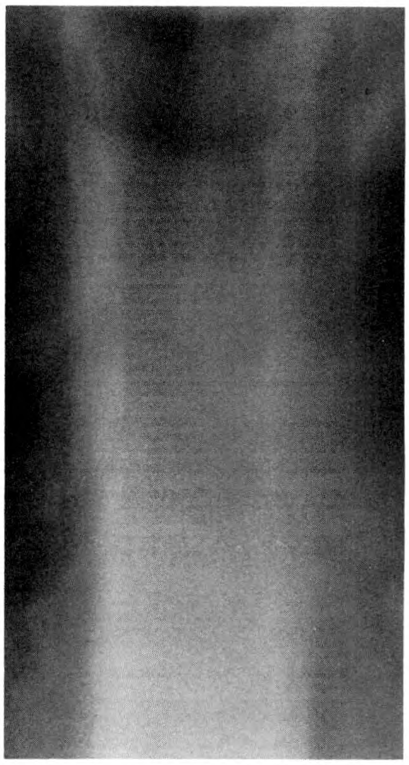

Fig 4. (Patient 2.) Tracheal tomography 12 months after slide tracheoplasty shows an adequate lumen throughout its length.

The technique of slide tracheoplasty requires the overlapping of the two divided stenotic segments. The potential limiting factor for this operation is the length of the tracheal stricture, but as 50% of the trachea can be resected [4], this operation can in theory be used when the entire trachea is narrowed. In our second infant, a severe stricture involving almost 60% of the entire tracheal length was repaired successfully. Other long-segment tracheal stenoses, for which resection may not now be appropriate, may become suitable for this procedure. If the tracheal stenosis is sufficiently localized, we still recommend segmental resection and reanastomosis without excessive circumferential dissection.

Preoperative assessment includes anteroposterior and lateral chest roentgenograms to assess the size of the infant's trachea. Tracheal tomography can delineate the severity of the stenosis. Computed tomography of the chest provides information on the tracheal stricture and any associated mediastinal or pulmonary abnormality. Right heart catheterization is necessary to exclude a pulmonary artery sling which may complicate the operation, as demonstrated in our first patient [8]. Computed tomography can be unreliable in this respect. Bronchoscopy and tracheoscopy defines the tracheal stenosis and bronchial anatomy. Limited tracheogram just before operation may be helpful in defining the distal anatomy but is potentially hazardous as it may precipitate total tracheal obstruction.

As stated by Grillo [9], successful tracheal reconstruction requires a flexible tube rigid enough to stay open and remain airtight. Dependable healing is also a prerequisite [9]. Native tracheal tissue was used in slide tracheoplasty. There was no loss of tracheal tissue with an increase in the reconstructed tracheal diameter, but the overall length was shortened. The trachea remained patent and stable, without stenting, as demonstrated in our second infant, who was extubated on the eighth postoperative day without any further ventilatory support. The transverse cervical approach was adequate, as more trachea lies in the cervical area of infants than in adults. This exposure provided good access, allowing accurate suturing and avoiding air leak or wound dehiscence. Our first infant had a right thoracotomy approach. He died of *Legionella pneumophilia* pneumonia acquired preoperatively, but there was no evidence of mediastinal air leak despite 5 weeks of extremely high ventilatory pressure. In both cases, tension at the anastomotic site was minimized with chin sutures to the sternal region for seven days postoperatively.

Maintenance of good gas exchange during the operation was essential. No extracorporeal circulation was required for cardiopulmonary support despite the critically narrowed airway. High-frequency jet ventilation with a small endotracheal tube into either lung has been used during the anastomosis, but we have found this less satisfactory. With the aid of a pulse oximeter to avoid prolonged periods of arterial desaturation, ventilation was continued by an endobronchial tube with intermittent tracheal/bronchial ventilation.

Grillo's [9] pioneering work on tracheal blood supply implies that our technique would fail due to tracheal devascularization. This was not the case, and we assume that the blood supply in the infant is different, at least in the funnel-shaped trachea, perhaps with greater intramural collaterals. Maeda and Grillo [10] also demonstrated the adequate growth of trachea after resection and anastomosis in puppies. However, in infants, even small or minimal anastomotic scarring may produce serious recurrent obstruction. Because native tracheal tissue was used in a slide tracheoplasty, with tension-free anastomosis, we hope early ingrowth of respiratory epithelium may minimize the risk of restenosis. Only time will tell whether, with continued growth of our patient, the tracheal lumen becomes inadequate. Despite the limited length of follow-up, the initial promising results make slide tracheoplasty a safe and effective technique for congenital funnel-shaped trachea in small infants.

We thank Dr J. O. Warner and Mr P. D. Bull, who allowed us to study their patients.

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Growth Potential of Latissimus Dorsi Muscle Flaps Used in the Cardiomyoplasty Procedure

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Synchronously stimulated latissimus dorsi muscles have been used for cardiomyoplasty in adult patients with heart failure. This procedure has the potential of being used in pediatric patients to enlarge a hypoplastic ventricle or to reinforce the right atrium in the Fontan procedure. The growth potential of such a myograft was studied. Young piglets weighing 18 to 22 kg underwent right latissimus dorsi cardiomyoplasty while the latissimus dorsi weight and myoplasty area were measured. In half of the animals, the myograft was not stimulated and in the others it was continuously stimulated with an atrioventricular sequential pacemaker to contract in synchrony with the heart. Two months later, with doubling of body weight, the hearts were removed for study. In both groups, significant ($p < 0.05$) increase in latissimus dorsi weight (+92% ± 15% nonpaced versus +77% ± 3% paced) and myoplasty area (+107% ± 13% nonpaced versus +116% ± 30% paced) were noted. The results in stimulated versus nonstimulated animals were not significantly different. Thus, not only does cardiomyoplasty offer a contractile tissue to repair or enlarge cardiac chambers, but this tissue can also grow in young patients and avoid subsequent size mismatch.

(Ann Thorac Surg 1989;48:636–8)

Dynamic cardiomyoplasty, in which a skeletal muscle transformed by chronic electrical stimulation to predominantly type 1 fatigue-resistant fibers [1] is used to replace or augment the myocardium, has been studied both experimentally [2] and clinically [3]. A synchronized burst stimulator has been developed to increase the efficiency of dynamic cardiomyoplasty [4]. Recent clinical experience indicates that in select patients, dynamic cardiomyoplasty can significantly augment the cardiac function of patients in severe heart failure [5]. One of the possible applications of this surgical technique is in the pediatric age group. Conceivably, a full-thickness cardiomyoplasty using autogenous pericardium as neoendocardium can enlarge a hypoplastic cardiac chamber, a stenotic outflow tract, or a major vessel [6]. Experimental studies are underway in several centers to use electrically paced skeletal muscle graft over the right atrium to improve blood flow through the lung after Fontan operation [7].

One of the attractive advantages with this approach of using autologous skeletal muscle graft in pediatric patients is its potential for growth. In contrast to prosthetic patch grafts, conduits, and valves, these muscle grafts may grow with the heart, avoiding repeated operations required to remedy the size mismatch as the patients grow. In the present study, we attempted to document such growth capability, and at the same time examined the question of whether, without electrical stimulation of the muscle graft, the repetitious passive stretch of the muscle by cardiac contractions alone could prevent its atrophy and sustain its normal growth.

Material and Methods

Eight Yorkshire piglets weighing between 18 and 22 kg were used. All the animals received humane care in compliance with the "Principles of Laboratory Animal Care." All piglets were intubated and anesthesia was maintained with intravenous pentobarbital and halothane (0.5% to 1.5%). A right latissimus dorsi muscle flap was developed by detaching the muscle at its tendinous insertion at the humerus and dividing it 15 to 20 cm distally. Care was taken not to injure the right thoracodorsal neurovascular pedicle throughout the entire procedure. Muscle flap weight was determined by suspending the muscle from a spring balance. The insertion of the muscle was then anchored to the chest wall while the bulk of the muscle mass was passed into the chest cavity through the bed of the resected third rib. The wound was then closed. After this, the heart was exposed through a median sternotomy. The pericardium was excised and the latissimus dorsi muscle was secured as an onlay flap over the pericardial surface of the right ventricle and right ventricular outflow tract in a rectangular pattern, using 6-0 Prolene sutures to anchor the skeletal muscle onto the myocardium. Hemoclips were placed at each corner. The surface area of the skeletal muscle in contact with the epicardium was determined as the "pregrowth" measurement. In half of these animals, the skeletal muscle flap was not electrically stimulated, whereas in the others a Medtronic model 7005 atrioventricular sequential pacemaker (Medtronic, Minneapolis, MN) was implanted. The ventricular lead (Medtronic model 5815) from the pace-

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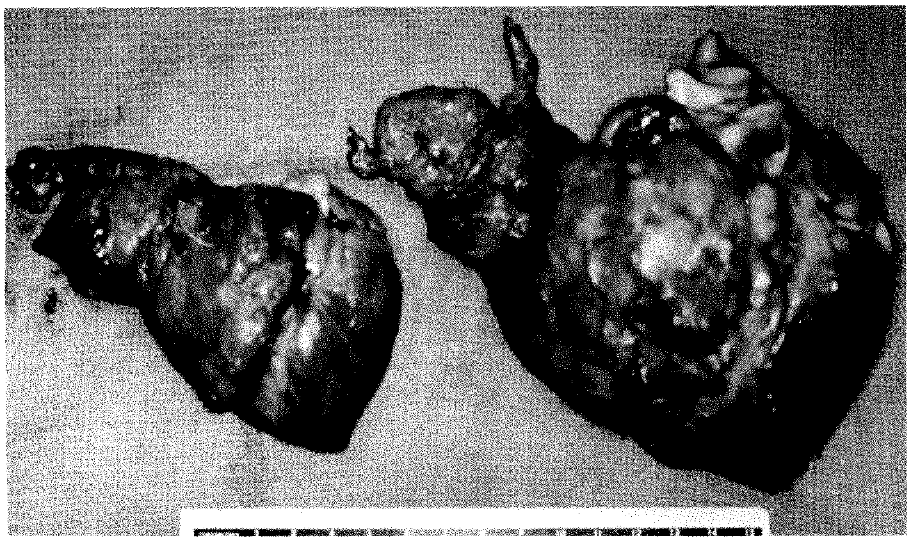

Fig 1. Specimens of porcine hearts with right-sided cardiomyoplasty taken from nonpaced animals (left) at the time of operation and (right) after growth has taken place. Note that both the heart and the latissimus dorsi muscle flap have grown.

maker was placed into the muscle mass in proximity to the thoracodorsal nerve, and the atrial lead (Medtronic model 6917) was attached to the right ventricle. The pacemaker output (maximum 6 V) was set to elicit visible contraction of the latissimus dorsi flap over the right ventricle. The pacemaker was activated intraoperatively and stimulation continued until the termination of the experiments. Synchronous 1:1 single-pulse stimulation of the skeletal muscle was therefore achieved in half of the experimental animals, and their subsequent growth was compared with that of the others, which only underwent passive stretch by the contractile action of the underlying cardiac muscle. Chest drains were inserted and the sternotomy was closed. Drains were removed after 24 hours.

The rotation of the latissimus dorsi muscle into the chest cavity caused no apparent impairment to the mobility and gait of the animals. The piglets were fed ad libitum and grew rapidly. They were killed after their body weight had doubled. This took 10 to 14 weeks. The muscle flap-heart complexes were removed en bloc and the latissimus dorsi flap was carefully dissected off the right ventricle and right ventricular outflow tract. The flaps were weighed and the suture area over the right side of the heart was measured using the nonabsorbable suture lines and the hemoclips at the corners as guides. Muscle flaps were histologically examined using hematoxylin and eosin stain, both to evaluate muscle fiber changes and to detect neovascularization at the myocardium-flap interface.

Results

There were 4 animals in each of two groups: paced and nonpaced. One animal in the paced group was excluded because of flap necrosis secondary to vascular compromise. Figure 1 demonstrates the gross appearance of the hearts at the time of the original operation and at the time of death, illustrating the obvious increase in size of both the muscle flap and the heart. Tables 1 and 2 present the data obtained in the growth study. The hearts weighed on the average 90 g at the onset of the experiment and reached an average weight of 225 g at the time of death.

In the paced piglets, the increase in flap weight was 77% ± 3%, and the suture area increased 116% ± 30% while the animals' body weight increased 120% ± 6%. Similar increases were seen in the nonpaced group, with corresponding figures of 92% ± 15%, 107% ± 13%, and 146% ± 20%, respectively. There were no statistically significant differences in any of these growth data between the paced and the nonpaced piglets (Student's nonpaired t test). Histology of the flaps showed no evidence of atrophy in both groups. We also failed to identify neovascularization at the interface in this model.

Table 1. Stimulated Myograft

Measurement	Animal Weight (kg)	Flap Weight (g)	Suture Area (cm^2)
Pre	21 ± 1	60.7 ± 1.4	10.4 ± 0.6
Post	46 ± 2	107.3 ± 2.3	22.8 ± 2.3
Percent increase	120 ± 6	77 ± 3	116 ± 30

Values expressed as mean ± standard error of the mean, n = 3. All changes significant at $p < 0.05$, paired t test.

Table 2. Nonstimulated Myograft

Measurement	Animal Weight (kg)	Flap Weight (g)	Suture Area (cm^2)
Pre	19 ± 1	60 ± 5.4	10.0 ± 1.3
Post	48 ± 3	112.5 ± 3.8	20.5 ± 1.9
Percent increase	146 ± 20	92 ± 15	107 ± 13

Values expressed as mean ± standard error of the mean, n = 4. All changes significant at $p < 0.05$, paired t test.

Comment

Attempts to use autologous skeletal muscle to repair or augment the myocardium go back to at least 1933 when Leriche [8] experimented with this concept, but in recent years it has gained renewed interest because of the increasing knowledge regarding muscle transformation [9] and the availability of suitable electrical stimulators [10]. Experimental studies on dynamic cardiomyoplasty have been brought to clinical applications in recent years, and their precise clinical indications and follow-up studies regarding their efficacy are currently being undertaken in a number of centers [3, 11]. Many investigators envision the application of this technique to pediatric patients with various congenital heart diseases. Yee and Ebert [12] in 1985 experimented with the use of anterior rectus myofascial island flaps to replace the left pulmonary artery, both as patch angioplasties and circumferential tube grafts. They were able to demonstrate that such muscles were nonthrombogenic, and no false aneurysm or degenerative changes occurred after 2 years. They, however, did not study the muscle flap growth quantitatively. Gaines and associates [13] in 1985 also grafted free gracilis flaps to the right ventricular outflow tract of swine, and showed that there was no aneurysmal dilatation at 10 weeks. They found, however, that although the muscle was viable, the flap was atrophic because of denervation. Currently, in a number of laboratories, studies are being conducted to augment the right atrial function after Fontan procedure [7]. In these studies, generally, muscle flaps with intact innervation are being used.

Our data clearly document the growth capability of such innervated pedicle flaps. Of interest is the lack of atrophy in the nonpaced group, which suggests that continuous passive stretch of these muscles by the action of the underlying myocardium can prevent disuse atrophy. This finding is consistent with those reported by Sola and colleagues previously [14]. We conclude, therefore, that cardiomyoplasty, either dynamic or adynamic (ie, nonpaced), can grow in pediatric patients, which adds another advantage to using the skeletal muscle either to replace or to augment the myocardium in such patients.

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Longer Survival After Resection of Non-Small Cell Lung Cancer in Japanese Women

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From 1974 to 1988, 492 patients (361 male and 131 female) with non-small cell lung cancer underwent pulmonary resection at our department. A retrospective study was done with special reference to the sex of the patients. There was no sex-related association with age distribution, operation, pathological T or N status, or staging. On the other hand, 77.9% of women had an adenocarcinoma and only 44.6% of men had this type of lesion. This was considered at least partly a result of sex difference in smoking habits. Women survived for a significantly longer time ($p = 0.0036$), and this difference could not be explained by differences in histological type. Presumably, hormonal factor(s) may contribute to the prognosis.

(Ann Thorac Surg 1989;48:639–42)

Lung cancer is on the increase in Japan. The age-adjusted death rate for men and women was 1.3 and 0.6 per 100,000 population in 1947 and 16.6 and 6.0 per 100,000 population in 1983, respectively [1]. These rates of increase in lung cancer mortality in Japan are very high as compared with findings in Western countries. It has been estimated that in 25 years Japan will have the highest mortality rate for lung cancer in the world [2].

Sex hormones have some relevance not only in cancers originating in sexual organs such as prostate, breast, or uterine endometrium but also in cancers of the esophagus [3, 4], stomach [5], or liver [6]. In this context, we analyzed various clinical characteristics in patients surgically treated for lung cancer with special reference to the sex of the patients and found that female sex was one of the favorable prognostic factors.

Material and Methods

During a 14-year period from February 1974 to March 1988, 590 patients underwent operation for treatment of bronchogenic carcinoma at the Department of Surgery II, Faculty of Medicine, Kyushu University, and their charts were reviewed with special reference to the sex of the patients. There were 556 patients with histologically proven non-small cell carcinoma of the lung and 34 with small cell lung cancer or low-grade malignancies. This study excluded 60 patients who underwent only exploratory thoracotomy and 4 who died within 30 days after operation. Thus the total number of the patients analyzed was 492 (361 male and 131 female).

Complete resection of the tumor by lobectomy or pneumonectomy combined with mediastinal lymph node dissection was performed whenever possible as standard treatment. Resected specimens were routinely examined pathologically for histological diagnosis and determination of the extent of lymph node spread. The postsurgical (pathological) stages were determined according to the TNM classification revised in 1986 [7].

Statistical comparison was made using the χ^2 test, survival rate was calculated by the Kaplan-Meier test, and the significance of the difference was analyzed by the generalized Wilcoxon test [8].

Results

Age

Age distribution of the patients is shown in Table 1. A peak incidence of lung cancer occurred in patients in the seventh decade in both sexes. The ratio of male to female patients was not dependent on age of the patients: 2.76:1 on the whole, 2.92:1 in patients aged less than 49 years, 2.73:1 in those aged 50 to 69 years, and 2.76:1 in those aged 70 years or older. The average age of female patients (61.9 years) did not differ significantly from that of male patients (62.3 years).

Smoking History

Distribution of the Brinkman index [9] (sum of the number of cigarettes smoked per day multiplied by years of smoking) is shown in Table 2. There was a striking difference in smoking habits between sexes. More than 75% of the women had never smoked, whereas only 5% of the men had never smoked. Fewer than 15% of female patients had a Brinkman index of more than 400, whereas the rate was 85.8% in male patients.

Histologic Types

Table 3 shows the distribution of histological type of tumors with respect to sex. There was a significant difference in histology between the sexes ($p < 0.01$). In male patients, squamous cell carcinoma and adenocarcinoma occurred in almost the same incidence, whereas about 80% of the women had an adenocarcinoma. When the incidence was compared in patients who had never

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Table 1. Age Distribution of Patients

Age (yr)	Male	Female
≤29	1	0
30–39	9	4
40–49	25	8
50–59	97	38
60–69	135	47
70–79	90	30
80–89	4	4

smoked, differences in histologic type were less evident between the sexes.

Type of Operation

The most frequent type of operation was lobectomy in both sexes. Pneumonectomy was performed for 18.8% of the men, a figure higher than that for women but with no statistical difference. This was probably due to male preponderance of squamous cell carcinoma, which tended to grow more centrally in the lung (Table 4).

T or N Status and Stages

There was no significant differences with respect to T or N status and stages between the sexes (Tables 5 and 6). Namely, the most common T or N status was T2 or N0, and about half of the patients had stage I disease.

Survival

Survival curves after pulmonary resection are shown in Figure 1. Female patients survived significantly longer after resection, as determined by the generalized Wilcoxon test ($p = 0.0036$). Table 7 shows survival rates stratified according to histological types of the tumor, stages, or Brinkman index. Women survived longer according to these stratifications as well, with the difference being significant ($p = 0.0234$) only in the case of stage III disease. When the survival rates for female or male patients were individually calculated according to histological types or Brinkman index as shown in Table 8, neither variable had a significant influence on the rates. Therefore, a favorable prognosis among female patients in lung cancer was not considered a result of the difference in histological types or Brinkman index between male and female patients.

Table 2. Brinkman Index

Brinkman Index	Male	Female
0	19 (5.3)[a]	99 (75.5)
1–400	32 (8.9)	13 (9.9)
401–800	121 (33.5)	11 (8.4)
801–1,200	114 (31.6)	6 (4.6)
1,201–1,600	35 (9.7)	0 (0.0)
1,601–2,000	16 (4.4)	1 (0.8)
≥2,001	20 (5.5)	0 (0.0)
Unknown	4 (1.1)	1 (0.8)

[a] Numbers in parentheses are percentages.

Table 3. Histological Types

Cancer	Male	Female
Adenocarcinoma	161 (15)[a]	102 (83)
Squamous cell	155 (2)	15 (6)
Large cell	38 (2)	10 (8)
Adenosquamous	7 (0)	4 (2)

[a] Numbers in parentheses are numbers of those who never smoked.

Comment

We have shown that the most prominent property that characterizes lung cancer in women was predominance of adenocarcinoma, and that this difference was at least partly attributable to sex difference in smoking habits. However, Lubin and Blot [10] reported that among those who had never smoked the ratio of incidence of adenocarcinoma to squamous cell carcinoma was significantly higher in women. A racial difference may also exist in the causal relationship between smoking and lung cancer. Hinds and colleagues [11] reported that only a minority of lung cancers could be attributed to smoking among Japanese and Chinese women in Hawaii as compared with native residents of Hawaii.

No characteristics were related to sex of the patients with respect to age distribution, operation, T or N status, and stage. The ratio of male to female patients was relatively constant irrespective of their age. This was in good contrast to the trend in occurrence of gastric cancer in Japan, in which this ratio increased significantly with age of the patients [12].

Many reports have described survival rate in lung cancer patients with respect to sex, and their results are quite conflicting. In a large series (8,807 patients) analyzed at the National Cancer Institute in 1961 in the United States, Ederer and Mersheimer [13] showed that the 5-year survival rate for surgically treated women with lung cancer was substantially greater than that for men. On the other hand, in an analysis of 3,834 patients in England, Bignall and Martin [14] reported that the survival rate of women was lower than that of men in all age groups in both the squamous and undifferentiated histological types, and in patients who underwent resection or radiotherapy and those who did not. Ashley and Davies

Table 4. Operation Performed

Operation	Male	Female
Lobectomy	250 (69.3)[a]	102 (77.9)
Bilobectomy	31 (8.6)	13 (9.9)
Pneumonectomy	68 (18.8)	13 (9.9)
Partial resection	12 (3.3)	3 (2.3)

[a] Numbers in parentheses are percentages.

Table 5. T and N Status

T or N Status	Male	Female	Total
T1	109 (30.2)[a]	50 (38.2)	159 (32.3)
T2	166 (46.0)	53 (40.4)	219 (44.5)
T3	59 (16.3)	14 (10.7)	73 (14.8)
T4	27 (7.5)	14 (10.7)	41 (8.4)
N0	230 (63.7)	80 (61.1)	310 (63.0)
N1	37 (10.2)	15 (11.5)	52 (10.6)
N2	92 (25.5)	35 (26.7)	127 (25.8)
N3	2 (0.6)	1 (0.7)	3 (0.6)

[a] Numbers in parentheses are percentages.

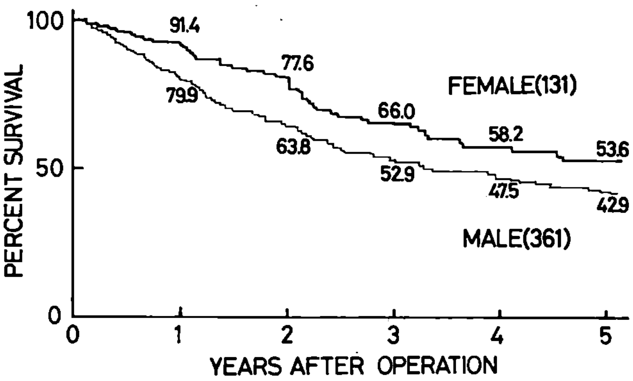

Fig 1. Survival rates of patients who underwent resection of the lung for cancer.

[15] reported that tumors in women tended to be less well differentiated and were less likely to be resectable than tumors in men. Harley [16] summarized these and other results, including his own, and reported that the prognosis for women with cancer of the lung was worse than for men in Wales, somewhat worse in England, possibly worse in France, and possibly better in the United States.

More recently, Kirsh and co-workers [17] of the University of Michigan reported that the survival of 78 women undergoing pulmonary resection and mediastinal lymph node dissection was considerably worse than that of men. They attributed these results to the predominance of adenocarcinoma in women and to the fact that about half of these women had stage III disease [17]. However, whether or not female sex itself influenced outcome was not considered. In a study dealing with only unresected cases in Japan, Sakurai and associates [18] showed that female sex was one of the most important factors linked to long-term survival.

In 492 patients, we found that survival time after resection of lung tumors was longer in women than in men. This was not due to differences in histology or smoking habits. The interaction between biological properties of the lung cancer cells and hormonal environment may be an explanation. Indeed, Chaudhuri and co-workers [19] reported that in cases of adenocarcinoma of the lung there was a significant incidence of receptors for estrogen (57%), as compared with findings in cases of squamous cell carcinoma of the lung (0%), and that the incidence of androgen receptor was equally low (14%). They also found that the cytosol receptor for any class of steroid was not detectable in small cell carcinoma of the lung. Hormonal factors were also suggested by an increased risk associated with late menopause and with a short menstrual cycle (less than 26 days) in an epidemiological study in China [20].

We suggest that sex-related factors independent of

Table 6. Stages

Stage	Male	Female	Total
I	181 (50.1)[a]	67 (51.1)	248 (50.4)
II	24 (6.6)	8 (6.1)	32 (6.5)
IIIA	109 (30.2)	31 (23.7)	140 (28.5)
IIIB	27 (7.5)	14 (10.7)	41 (8.3)
IV	20 (5.6)	11 (8.4)	31 (6.3)

[a] Numbers in parentheses are percentages.

Table 7. Survival Rates According to Histological Types, Stages, and Brinkman Index

	Year					
Variable	1	2	3	4	5	p Value
Adenocarcinoma						
M	79.9	63.8	52.9	47.5	42.9	0.0924
F	91.4	77.6	66.0	58.2	53.6	
Squamous cell carcinoma						
M	78.5	61.0	52.7	49.8	47.6	0.5156
F	86.7	79.4	79.4	66.2	39.7	
Stage I						
M	94.2	83.4	72.7	67.7	62.1	0.1500
F	93.9	92.4	86.5	77.5	74.8	
Stage II						
M	69.6	47.8	34.2	18.3	18.3	0.1836
F	100	75.0	60.0	60.0	20.0	
Stage III (IIIA + IIIB)						
M	67.3	45.3	34.6	30.1	24.9	0.0234
F	86.6	52.7	33.4	26.7	26.7	
BI = 0						
M	94.7	83.6	58.5	50.2	41.8	0.4360
F	94.8	80.4	66.3	56.1	54.2	
BI = 1–400						
M	71.9	43.5	40.2	35.7	35.7	0.2301
F	76.9	76.9	61.5	61.5	41.0	
BI > 400						
M	80.8	65.7	54.5	48.8	44.0	0.3767
F	83.3	66.7	66.7	66.7	57.1	

BI = Brinkman index.

Table 8. Survival Rates in Male and Female Patients According to Histological Type or Brinkman Index

	Year					p-Value
Variable	1	2	3	4	5	
Female patients						
Adenocarcinoma	94.0	77.6	62.9	55.8	53.8	0.7535
Squamous cell carcinoma	86.7	79.4	79.4	66.2	39.4	
BI = 0	94.8	80.4	66.3	56.1	54.2	0.7995
BI = 1–400	76.9	76.9	61.5	61.5	41.0	
BI ≥ 401	83.3	72.2	66.7	66.7	57.1	
Male patients						
Adenocarcinoma	86.6	69.1	57.0	48.5	42.1	0.3832
Squamous cell carcinoma	78.5	61.0	52.7	49.7	47.6	
BI = 0	94.7	83.6	58.9	50.5	42.1	0.1780
BI = 1–400	71.9	43.5	40.2	35.7	35.7	
BI ≥ 401	80.8	65.7	54.5	48.8	44.0	

BI = Brinkman index.

other factors may be linked to the more favorable prognosis of women with lung cancer. It is tempting to think that hormone therapy could be applicable for control of lung cancer.

We thank M. Ohara for comments.

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Safety of Metallic Surgical Clips in Patients Undergoing High-Field-Strength Magnetic Resonance Imaging

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Use of metallic clips with ferromagnetic properties in patients undergoing a large variety of surgical procedures, and in particular, in coronary artery and other vascular reconstructions, has become increasingly popular. The safety of these commonly used surgical clips when subjected to high-intensity diagnostic magnetic resonance imaging fields is still debated. Commonly used hemostatic metallic clips manufactured by the Weck and US Surgical Corporations were tested in an in vitro system to assess their safety with regard to migration and displacement. The two tested hemostatic metallic clips proved safe and did not migrate or become dislodged when they underwent magnetic resonance imaging scans. This is in direct contrast to multiple cerebral aneurysm clips, also tested, which have highly ferromagnetic properties and were previously shown to migrate with disastrous results in patients undergoing diagnostic magnetic resonance imaging scans. This study supports the continued widespread use of small metallic hemostatic clips in the myriad of procedures in which they are presently used and illustrates the need for methods of evaluating such devices before they are clinically implanted.

(Ann Thorac Surg 1989;48:643–5)

Use of metallic clips with ferromagnetic properties in patients undergoing a great variety of surgical procedures, such as in coronary artery revascularization for vein side branch ligation or as implantable radiographic markers, has become very widespread. The potential for clip displacement, vascular erosion, migration, or embolization of such ferromagnetic biomedical implants has been considered an absolute or relative contraindication for postoperative diagnostic magnetic resonance imaging, particularly when high field strengths are used. Good substitutes for small metallic surgical clips are not readily available. Because the widespread use of such clips could therefore interfere with subsequent important diagnostic imaging of patients, we performed an in vitro study to determine the potential for migration or displacement of two types of commonly used surgical clips in a biological medium subjected to high-field-strength magnetic resonance imaging systems.

Material and Methods

Two different types (USSC Auto Suture Titanum 132861, United States Surgical, Norwalk, CT, and Weck Tantalum Hemiclip 523100, Weck, Princeton, NJ) of medium-sized metallic clips were selected. Multiple clips were imbedded in a standard blood agar medium that was maintained at 37°C and allowed to gel. Clips in multiple orientations and with different degrees of closure (open, half closed, and closed) were placed in the agar medium. The magnetic clips, although not completely visible, were photographed with routine radiological techniques to obtain a direct image of the position of all the magnetic clips in the Petri dishes (Fig 1).

Two different cerebral aneurysm clips with highly ferromagnetic properties were selected as a control to demonstrate the sensitivity of the technique. Six Scoville aneurysm clips (model 143-430) and six Mayfield aneurysm clips (model 200-101) were imbedded in an identical 37°C blood agar medium and subjected to similar x-ray photography.

The blood agar medium containing the metallic clips was then subjected to 45 minutes of 1.5 T magnetic resonance imaging using a General Electric magnetic resonance imaging scanner. Next, the agar plates were x-rayed again, and the two films were compared. The plates were also photographed conventionally using transmitted light to look for tracks or other agar deformities created in the medium due to migration of the clips. All films were reviewed by two independent radiologists and compared.

Results

All observers agreed that the premagnetic field and postmagnetic field x-ray films of the small hemostatic clips were completely superimposable with no evidence of rotation or migration of any of the clips (Fig 2). The photographs of the transluminated agar medium revealed no tracks, pockets, or artifacts that could suggest any otherwise unrecognized motion or displacement of the position of the metallic clips.

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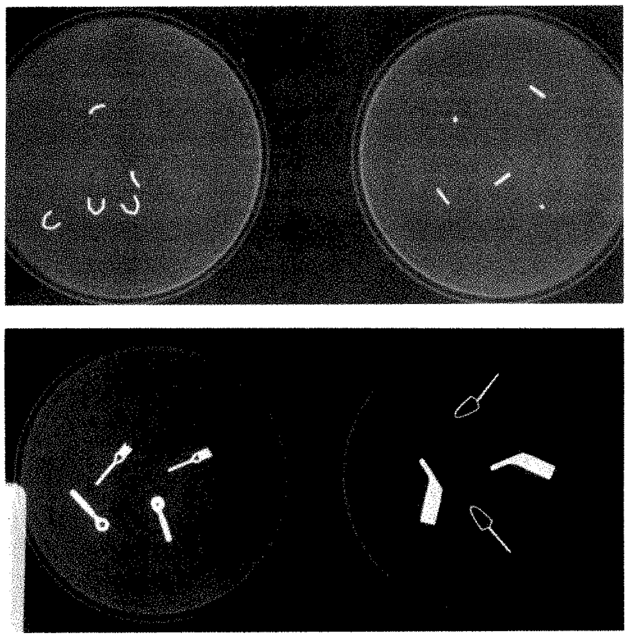

Fig 1. Radiograph of metallic clips before period of magnetic field exposure (left top, Weck; left bottom, US Surgical; right top, Scoville; right bottom, Mayfield).

The observers also agreed that the premagnetic field and postmagnetic field x-ray films of the cerebral aneurysm clips were nonsuperimposable and showed evidence of severe displacement and rotational forces on every clip in each agar dish. The transmission light photography likewise revealed multiple tracks and pockets consistent with displacements measuring as much as 3 to 4 cm. The planes of the clips, as well as the locations of the clips, were all markedly altered.

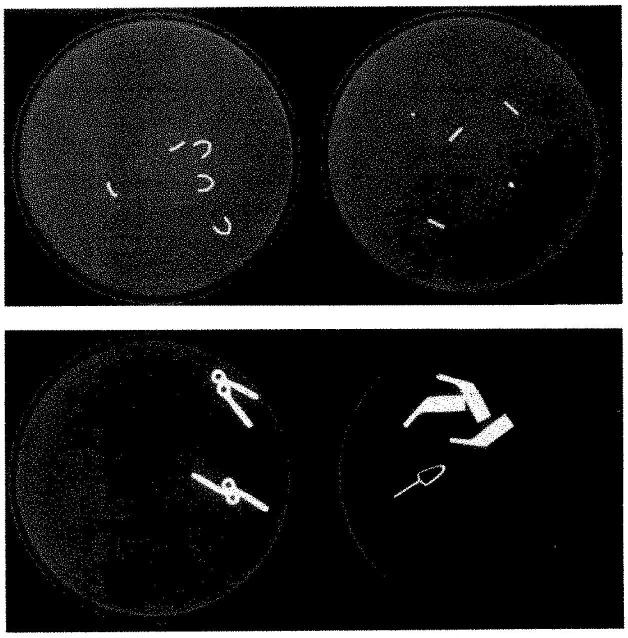

Fig 2. Radiograph of metallic clips after the period of magnetic field exposure (left top, Weck; left bottom, US Surgical; right top, Scoville; right bottom, Mayfield).

Comment

The principle danger of magnetic resonance imaging in patients who have undergone surgical procedures using ferromagnetic biomedical implants is associated with displacement of the metallic object [1–11]. The gradient of the strength of the magnetic field, the degree of ferromagnetism of the metallic object, the location and orientation of the object, and the length of time the object has been implanted all serve to determine the risk of migration. Although hemostatic clips are relatively small, they are frequently placed in strategic locations near large vessels and other important structures, making the potential for serious damage great. The frequent use of clips for resecting and marking neoplasms, as well as in multiple types of cardiovascular, peripheral vascular, and cerebrovascular surgery, might complicate the need for subsequent magnetic resonance imaging as an important diagnostic postoperative modality.

Migration of large ferromagnetic objects such as orthopedic prosthesis or, indeed, metallic cerebral aneurysm clips has been well documented. In one study the migration of aneurysm clips in laboratory animals was of magnitude sufficient for the deflection forces to remove the aneurysm clips from the blood vessels to which they had been applied [1].

The motion of the ferromagnetic cerebral aneurysm clips in these blood agar plates demonstrates the potential dangers of the use of such devices when placed in proximity to major vascular or neurological structures and substantiates the sensitivity of the blood agar medium as an appropriate testing means. This confirms the potential for serious complications should clips with equivalent ferromagnetic properties be applied for hemostatic reasons at the time of a surgical procedure.

The determination of the complete absence of migration, rotation, or displacement of small hemostatic metallic clips is reassuring, demonstrating the safety of obtaining a magnetic resonance imaging scan in patients who have previously undergone metallic clip implantation. This absence of hazard associated with small hemostatic clips has been confirmed in other studies as well, using a very different static magnetic displacement system [12].

Although the two tested hemostatic vascular clips do not demonstrate clinically significant ferromagnetic properties when embedded in physiological medium, the potential for the development and use of other hemostatic clip devices or other metallic implants with greater ferromagnetic properties remains. We therefore suggest not only that the hemostatic and mechanical properties of any such device be fully evaluated before it is used, but that the presence or absence of any substantial ferromagnetic properties also be fully evaluated. Only in this way will the potential catastrophe of migration or displacement be avoided.

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Evaluation of Arterialized Vein Graft Permeability With Evans Blue Dye and Iodine 125-Labeled Albumin

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This study measured endothelial permeability, quantitatively (with iodine 125-labeled albumin) and qualitatively (with Evans blue dye) in arterialized vein grafts in 9 adult dogs. In each dog, the right common carotid artery was ligated and arterial flow was reconstituted with a 5-cm bypass graft of right external jugular vein. Twenty-four hours before the dogs were killed, each dog received both ^{125}I-labeled albumin (8 μCi/kg) and Evans blue dye (15 mg/kg) intravenously. After the dogs' death, each arterialized vein graft and each left external jugular vein (as control) was harvested and studied. Arterialized vein graft permeability was significantly increased over control at 1, 3, and 6 months (3 dogs at each interval). Scanning electron microscopy revealed confluent endothelium in all specimens. This increased permeability may play a role in vein graft atherosclerosis.

(Ann Thorac Surg 1989;48:646–50)

Numerous clinical studies have shown that the autologous vein grafts routinely used to bypass obstructing lesions of the coronary arterial tree are prone to development of atherosclerosis [1–12]. Although most of these atherosclerotic lesions occur more than 5 years after implantation, there has been a report [3] of gross atherosclerotic lesions occurring less than 6.5 months after operation. Microscopic evidence of atherosclerotic changes has been found within 3 months after implantation [12]. This has led surgeons to prefer using internal mammary artery as a bypass conduit because of its "resistance" to development of atherosclerosis [6]. Because most coronary artery bypass procedures still require use of autologous vein grafts, however, vein graft atherosclerosis continues to be a clinical problem.

The cause of accelerated vein graft atherosclerosis is unknown. Previous experimental work with arterial atherosclerosis has suggested that increased endothelial permeability may play a central role [13, 14]. Studies in various animal species have shown that atherosclerotic lesions tend to develop in regions of the arterial tree that have increased endothelial permeability, as measured by uptake of albumin labeled radioactively and with Evans blue dye (EBD) [13–17]. Because endothelial permeability of arterialized grafts has not been studied, we used iodine 125-labeled albumin and EBD to evaluate permeability of arterialized vein grafts in dogs over a 6-month period.

Material and Methods

In 9 adult mongrel dogs (19 to 30 kg) general anesthesia was induced with pentobarbital (30 mg/kg intravenously), an anterior vertical neck incision was made, and the right common carotid artery and right external jugular vein were dissected out along their entire length. After systemic heparinization (100 U/kg intravenously), a 5-cm segment of the right external jugular vein was excised and gently irrigated with heparinized (10 U/L) Ringer's lactate solution. Only the end of the vein was handled and only with fine vascular forceps. The common carotid artery was doubly ligated with 1-0 silk sutures, and arterial flow was reestablished with the excised right external jugular vein by performing two end-to-side anastomoses with 6-0 Prolene (Ethicon; Somerville, NJ). At 1, 3, and 6 months after operation 3 dogs were killed (with 26% sodium pentobarbital/10% isopropyl alcohol/20% propylene glycol [Sleepaway; Fort Dodge Lab Inc, Fort Dodge, IA], 10 mL intravenously). Twenty-four hours before the dogs were killed, each dog received both ^{125}I-labeled canine albumin (8 μCi/kg, Sigma Chemical, St. Louis, MO) and EBD (15 mg/kg, Harvey Laboratories) intravenously.

At death each dog's arterialized vein graft and left external jugular vein (as control) were harvested. Each vessel was opened vertically, and its intimal surface was photographed for qualitative assessment of EBD uptake. A central 1-cm section of each graft was excised for scanning electron microscopy. The remaining parts of each vessel were then sectioned into four equal fragments. Each fragment was weighed and scanned with a γ-counter for measurement of ^{125}I uptake (counts per gram per five minutes). A mean uptake was calculated for each vessel.

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Table 1. Uptake of ^{125}I-Labeled Albumin in Arterialized Vein Graft and Control Vein

		Uptake (counts/g/5 min)		
Interval	Dog	Graft	Control	Δ
1 mo	1	8.5	6.1	2.4
	2	7.2	0.7	6.5
	3	5.5	2.5	3.0
	Mean ± SD			3.97 ± 2.21[a]
3 mo	4	7.6	2.0	5.6
	5	8.0	1.1	6.9
	6	5.5	1.9	3.6
	Mean ± SD			5.37 ± 1.66[a]
6 mo	7	4.4	0.6	3.8
	8	5.8	0.5	5.3
	9	3.0	1.1	1.9
	Mean ± SD			3.77 ± 1.70[a]

[a] $p < 0.05$, difference between graft and control.

Δ = difference between graft and control; SD = standard deviation.

For each dog the difference between the graft and the control vessel was calculated. Statistical analysis for each group of dogs was performed with a one-sample (paired), one-tailed *t* test. An average percentage increase in counts of the graft over the control with a range was calculated for each group.

For scanning electron microscopy the specimens were fixed for one hour in Trump's fixative and then rinsed twice in 0.1 M phosphate buffer (pH 7.2). The tissues were dehydrated in graded alcohol (60%, 70%, 80%, 95%, and 100%), placed in perforated BEEM (Better Equipment for Electron Microscopy) capsules, and immersed again in 100% alcohol. After drying in a Polaron critical-point dryer, the specimens were mounted on aluminum stubs with colloidal graphite, coated with gold/palladium, and examined with the Etec autoscan scanning electron microscope at 20 kV.

All procedures used in this study were conducted in accordance with institutional guidelines concerning the care and use of experimental animals.

Results

^{125}I-Labeled Albumin Uptake

In all 9 dogs, the arterialized graft had greater uptake of ^{125}I-labeled albumin than did the control vessel (Table 1). For each group of dogs, the difference between graft and control was statistically significant. The mean increase in albumin uptake for arterialized graft over control vessels was 331% (range, 39% to 930%) at 1 month, 367% (190% to 627%) at 3 months, and 621% (170% to 1,060%) at 6 months. Because of the small numbers of dogs and wide range within each group, no conclusion could be drawn as to whether albumin uptake was truly increasing as a function of time.

Evans Blue Dye Uptake

The permeability of the arterialized vein graft endothelium was confirmed qualitatively with EBD. All nine arterialized vein grafts stained more heavily than did the control vessels. Examples of graft and control vessels from 1 dog from each of the three groups are shown in Figure 1.

Scanning Electron Microscopy

Scanning electron microscopy performed on a section of each dog's arterialized vein graft revealed confluent endothelium covering the intimal surface of each vessel (Fig 2).

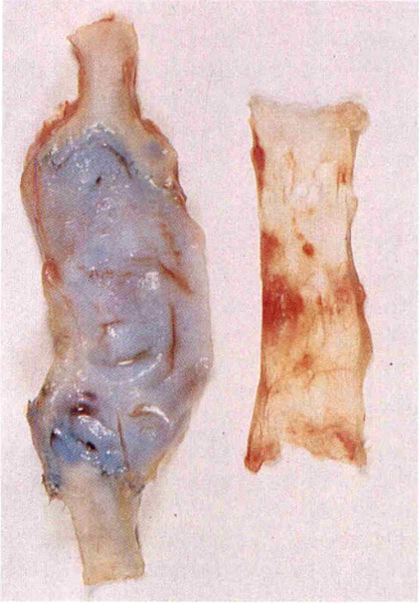
Comment

In 1961, Ejrup and colleagues [1] first reported the occurrence of atherosclerosis in an arterialized vein graft (femoropopliteal bypass). Not until 1970 did Beebe and coworkers [2] report the second case of vein graft atherosclerosis, also in a femoropopliteal bypass graft. Both groups of investigators concluded that vein graft atherosclerosis is uncommon when autologous vein grafts are used for femoropopliteal reconstruction.

In the early 1970s, evaluation of arterialized vein grafts removed during coronary artery bypass operations demonstrated that atherosclerosis also developed in veins used to bypass coronary artery obstructions. Barboriak and associates [3] discovered atherosclerotic changes in vein grafts of 6 of 8 patients undergoing reoperation 6 to 61 months after their first operation. In a morphological review of 97 saphenous vein coronary artery bypass grafts, Bulkley and Hutchins [5] reported nonocclusive concentric intimal fibrous plaques in all patent grafts at more than 30 days after implantation; the lesion was believed to be indistinguishable morphologically from the fibrous plaques of arterial atherosclerosis. A subsequent anatomical study of 99 vein grafts recovered at autopsy from 55 patients revealed true atherosclerotic lesions in 3 (12%) of 26 vein grafts from normolipemic and 11 (79%) of 14 vein grafts from hyperlipemic patients who had survived for 13 to 75 months after aortocoronary bypass [11]. None of the 59 grafts in place less than 12 months had atherosclerotic lesions.

Serial angiographic postoperative studies of coronary artery bypass grafts strongly suggest that vein graft atherosclerosis is the main threat to the long-term patency of the graft. Using angiography, FitzGibbon and colleagues [9] demonstrated intimal irregularities compatible with atherosclerosis in 9% of vein grafts at 1 year, increasing to 42% of vein grafts at 5 years after operation. Campeau and co-workers [10] also reported angiographic evidence of atherosclerosis in 45% of patent vein grafts studied 7 to 12 years postoperatively. If the rate of "new graft closures" (26%) found at 7 to 12 years after operation is included, atherosclerosis is present in more than 70% of vein grafts at this interval after operation. These investigators [10] also reported that, if only new lesions are considered, atherosclerosis develops at an accelerated pace in vein grafts as compared with native coronary arteries.

The cause of the accelerated vein graft atherosclerosis is unknown. Previous research with EBD strongly sug-



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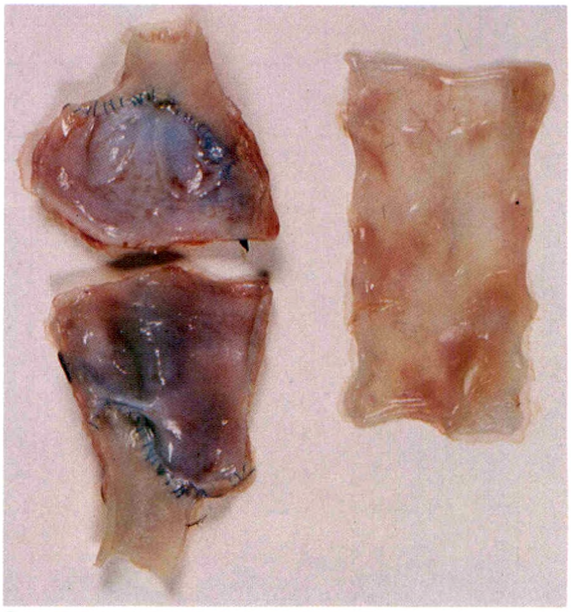

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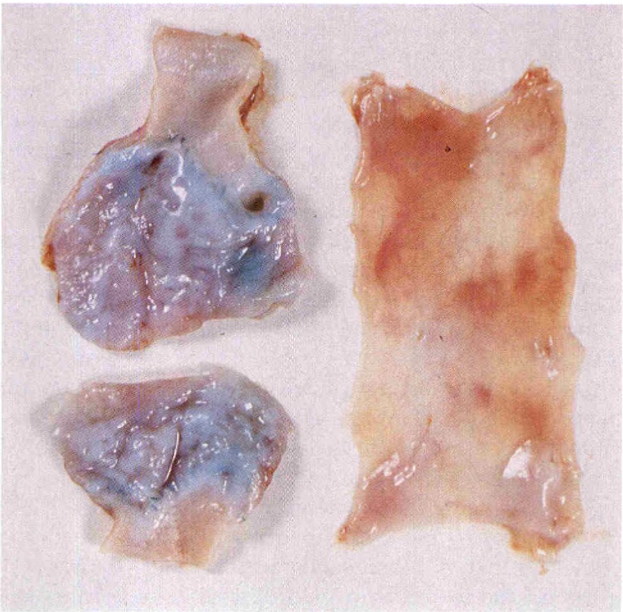

B

Fig 1. Intimal surfaces of arterialized vein grafts (Left) and respective control (Right) at 1 month (A), 3 months (B), and 6 months (C) after operation. Marked blue staining of the arterialized vein grafts by Evan's blue dye as opposed to the control veins is apparent. (A central section of vein graft was removed for scanning electron microscopy before photography in B and C.)

gested that increased endothelial permeability may play an important role in the pathophysiology of arterial atherosclerosis [13, 14]. When administered intravenously to an animal, EBD forms a strong bond to albumin and accumulates in the intima and adjacent medial tissue layers of arteries; thus it can be used to measure the permeability of the arterial wall to plasma protein [15–17]. Systemic administration of EBD produces a blue staining of the intima that tends to vary from point to point along the arterial tree in a characteristic topographic pattern. When normal animals are fed an atherogenic diet to increase serum cholesterol concentration, the topographic distribution of the resulting sudanophilic lesions is strikingly similar to the pattern of blue-stained tissue in normal animals after systemic administration of EBD. This finding has led to the conclusion that increased endothelial permeability may play a central role in the genesis of arterial atherosclerosis.

Our study shows that the endothelial permeability of a vein increases significantly when it is transplanted into the arterial circulation. Increases in permeability in the first few months could be due to endothelial denudation or endothelial dysfunction, or both. In dogs, Wyatt and Taylor [18] reported that vein grafts were only 50% covered with endothelium at 2 weeks after transplantation; this increased to 80% to 90% at 4 weeks and to 100% at 6 to 8 weeks after operation. Although our scanning electron micrographs did not show any endothelial denudation at 1 month, small areas of endothelial denudation could have been missed, and they may have played some role in increased vein graft permeability early after operation (1 month). However, Wyatt and Taylor's find-

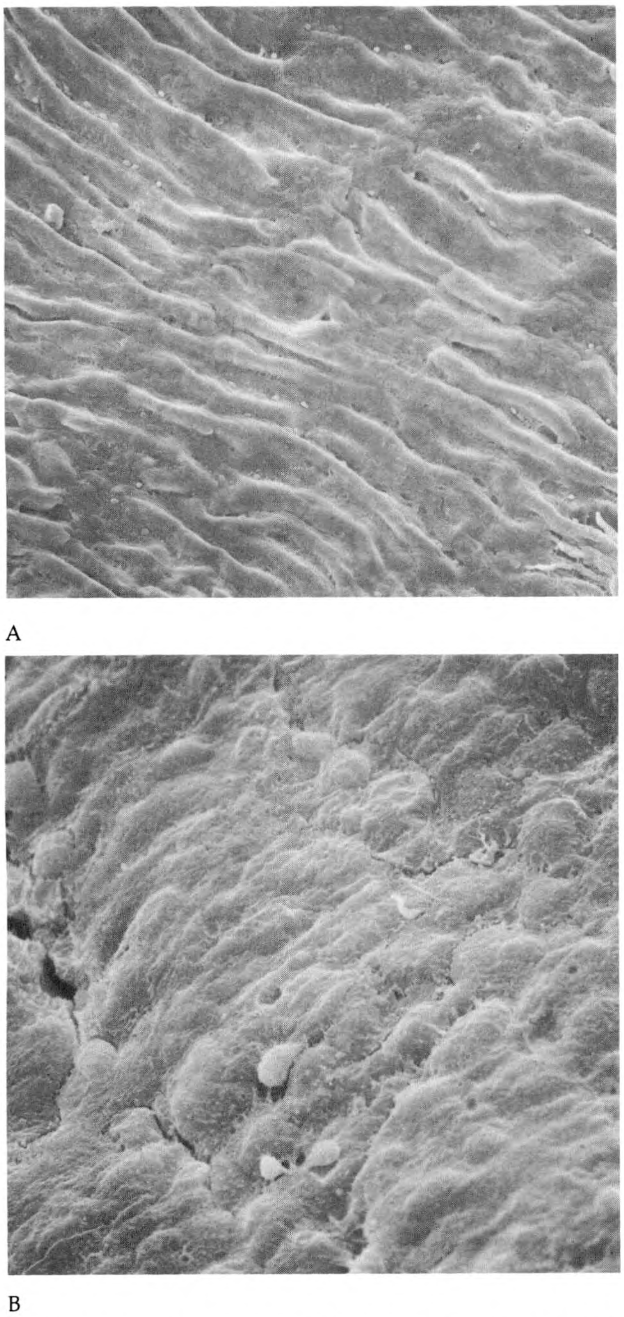

Fig 2. Scanning electron micrographs of luminal surfaces of arterialized vein grafts at 1 month (A) and 3 months (B) after operation, demonstrating confluence of endothelium. (A, ×200; B, ×1,000; both before 9% reduction.)

ing of a complete endothelial lining at 6 to 8 weeks and our demonstration of a confluent endothelial layer at 3 and 6 months would discredit endothelial denudation as an explanation for the increased endothelial permeability noted at 3 and 6 months.

The finding of increased endothelial permeability in the presence of confluent endothelium suggests that arterialized vein graft endothelium does not accommodate to its new high-pressure pulsatile environment, at least during the 6 months after implantation. This endothelium is less effective as a barrier to the passage of macromolecules into its wall, which may be one explanation for accelerated vein graft atherosclerosis. The reason for this increased permeability either across or between endothelial cells is unknown. The endothelial lining of large arterial vessels has been shown to be able to release prostaglandins [19], to degrade bradykinin and generate angiotensin II [20], and to synthesize histamine [21], all of which have been implicated in the control of vascular permeability.

Canine arterial endothelium normally produces larger quantities of prostacyclin than does venous endothelium [22, 23]. Because transposition of a vein into the arterial system does not produce any increase in venous endothelium prostacyclin production [24], the inherent biochemical differences between venous and arterial endothelium are not altered by transposition of a vein into the arterial system. Prostaglandins can prevent a mediator-induced increase in endothelial permeability [25–27]. Thus, the inability of venous endothelium to alter biochemical production of certain compounds such as prostaglandins after transposition into the arterial system may play a role in the increased endothelial permeability of arterialized vein grafts.

Another explanation for this increased permeability may involve the intercellular junction. In a study using freeze-cleaned preparations from rats, Simionescu and associates [28] found differences between the attachments in arterial and venous endothelial cells. Although the endothelial cells of the arterial and venous systems have the same type of intercellular junctions (occluding and communicating), the attachments appeared to be much "tighter" in arteries than in veins. Hypertension has also been demonstrated to increase the permeability of arterial intercellular junctions [29, 30]. Thus, increased permeability of the arterialized vein graft may be occurring at the intercellular junction.

This study has demonstrated a significant increase in venous endothelial permeability at 1, 3, and 6 months after transposition of a vein into the arterial tree. In view of the previous association between increased arterial endothelial permeability and atherosclerosis, increased permeability of an arterialized vein graft may play a role in the pathogenesis of vein graft atherosclerosis. If this is true, further research should be undertaken to improve the barrier function of arterialized vein graft endothelium (ie, to prevent an increase in endothelial permeability) to forestall development of atherosclerosis in it.

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Thoracoscopic Ablation of Blebs in the Treatment of Recurrent or Persistent Spontaneous Pneumothorax

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Recurrent (6 patients) or persistent (4 patients) spontaneous pneumothorax was treated with thoracoscopic ablation of blebs using an electrocautery as an alternative to lung resection. Patients' ages ranged from 17 to 39 years (mean age, 29 years). Ablation was successful in 9 but failed in 1. The mean postoperative hospital stay of the successful cases was 2.1 days. The only failure in this series had an extensive bullalike air cyst involving the apex, and cauterization could not ablate it completely. The follow-up period ranged from 2 to 22 months. During this period, no recurrence was noted.

(Ann Thorac Surg 1989;48:651–3)

Spontaneous pneumothorax is one of the most common diseases in the field of thoracic surgery. It occurs without any warning in otherwise healthy individuals [1–4]. The most common cause is a ruptured bleb, which occurs only in young patients. Occasionally, rupture of a bulla, bronchogenic carcinoma, or granuloma is the cause of spontaneous pneumothorax. This type is common in older persons. In principle, the first episode is treated with a tube thoracostomy, which is successful in controlling air leak within several days in approximately 80% of the patients. However, recurrence is not uncommon [5]. In the rest of the patients, air leak persists for days. These patients require some form of intervention such as pleurodesis [5–7] or partial resection of the lungs with or without pleurectomy [8–13].

In 1971, I decided to cauterize these blebs through a thoracoscope to seal off the hole and to cauterize all unruptured blebs to prevent recurrence. There were two questions to be answered: whether the cauterization could seal the air leak and whether the recurrence could be prevented by cauterizing the unruptured blebs.

I first tested this method during thoracotomy in a patient with recurrent pneumothorax. Before resecting the apex of the lung, I cauterized the blebs. The heat created by cauterization instantly made the blebs shrink, and the method appeared to work. I used an old thoracoscope of Jacobaeus [14] in an 18-year-old woman with recurrent spontaneous pneumothorax. The air leak stopped in three days, and she made an uneventful recovery. Subsequently, 4 other young patients with recurrent spontaneous pneumothorax were treated successfully by this technique. The question of whether this technique could prevent recurrence was not answered at that time.

This procedure, thoracoscopic ablation of the blebs, was not performed in the following 16 years for no obvious reason other than the fact that the old scope had been lost. During this period, however, I continued to perform thoracoscopy for diagnostic purposes. The experience gained during diagnostic thoracoscopy and improved instrumentation and anesthesia technique prompted me to restart this procedure in 1987. The present study is a summary of this renewed series.

Patients and Methods

The age and sex distribution of the patients is shown in Table 1. All patients were informed of the experimental nature of this procedure and a possible necessity of subsequent thoracotomy if the procedure failed. The patients ranged in age from 17 to 37 years, and there were equal numbers of female and male patients. Two patients (patients 1 and 6) had a history of spontaneous pneumothorax on the opposite side, including thoracotomy (patient 6). One patient (patient 5) had simultaneous bilateral spontaneous pneumothorax 48 hours after bilateral breast implant operation and was treated with bilateral thoracoscopies. Patient 9, a 27-year-old woman, had three spontaneous pneumothoraces within 18 months. She had multiple sclerosis and right-sided hemiparesis. In all 6 patients, recurrent spontaneous pneumothorax occurred within 6 months after the previous episode. Four patients had a first episode of spontaneous pneumothorax, but air leak persisted for more than five days.

Instruments

A glass-rod lens (Storz, Tuttlingen, FRG) scope, a suction/coagulator, and a grasping bipolar forceps-coagulator were used in all patients in this series.

Procedure

Double-lumen endotracheal intubation facilitated the procedure. A pulse oximeter was used to monitor the patient. The patient was placed in a decubitus position. Two small stab wounds were made in the skin at the third intercostal

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Table 1. Outcome of Thoracoscopic Ablation of Spontaneous Pneumothorax in 10 Patients

Patient No.	Age (yr)	Sex	Side	Cause	Result
1	29	M	R	Recurrent	Success
2	17	F	L	Recurrent	Success
3	28	M	R	Persistent	Success
4	24	M	L	Recurrent	Success
5	32	F	R	Persistent	Success
6	37	F	L	Recurrent	Success
7	31	M	R	Recurrent	Success
8	26	F	R	Persistent	Success
9	27	F	R	Recurrent	Failure[a]
10	21	M	R	Persistent	Success

[a] Huge bullous pseudopneumocyst involving the apex could not be completely ablated with an electrocautery and required wedge resection of the apex four days later.

space and the midclavicular and at the fourth intercostal space and the anterior axillary line. The chest wall was entered with a trocar. One-lung ventilation was started to collapse the lung. Any fibrous adhesion noted was cut with a coagulator and any pleural effusion was aspirated. The lungs had to be freed completely from the chest wall and collapsed to obtain satisfactory visualization of the lungs. The apex of the upper lobe, the anterior edge of the anterior segment of the upper lobe, and the edge of the superior segment of the lower lobe were inspected. If visualization of the lower lobe was unsatisfactory, another stab wound was made at the fifth intercostal space. A ruptured bleb itself was not identifiable as a hole because the lung was collapsed. A freshly ruptured bleb (ruptured within two days) was frequently surrounded by hemorrhage. An old ruptured bleb (ruptured for several days) was always covered with whitish-yellowish fibrin materials. When these hemorrhagic spots or fibrin deposits were identified they were grasped with the coagulator forceps and a coagulation energy at 30 to 35 W-s was applied until the tissue turned whitish and shrank. In contrast, unruptured blebs were easily identified as hemispherical cysts, even when the lungs were completely collapsed. All unruptured blebs were coagulated with the suction/coagulator metal tube. After all visible blebs were coagulated, a 20F chest tube was inserted through one of the stab wounds and connected with a pleural suction device. The other stab wound was closed with stitches.

Results

Results of the procedure, which was successful in all but 1 patient (patient 9), are shown in Table 1. In successful cases, the air leak from the chest tube stopped within two days. The mean postoperative hospital stay of the successful cases was 2.1 days. This procedure failed in 1 patient who subsequently underwent a thoracotomy five days later. The extensive bullous changes involving almost the entire apical segment were noted during operation. The follow-up period of 10 patients in this series varied from 2 to 22 months. During this follow-up period there was no recurrence.

Comment

Thoracoscopy, originally described by Jacobaeus [14] in 1915, was widely used during the tuberculosis era but is seldom used today. I became interested in this technique and used it extensively for diagnostic purposes. This experience formed the basis for developing therapeutic usage as described here. Waterman briefly described a similar technique in a discussion of Crenshaw's publication [15] in 1950. It was impressive to observe that the air leak stopped very quickly after electrocoagulation of the leaking ruptured bleb. The tissue coagulation probably produced a welding effect on the protein, sealing the air leak. Success of this procedure may depend on the welding effects of heat applied to the blebs; the one failure in this series could have been prevented had better welding means been used. LoCicero and colleagues [16–18] reported that the carbon dioxide laser was more effective than electrocauterization in sealing air leaks from the lungs. This modality was tested in a 25-year-old woman with persistent pneumothorax. In addition to ruptured blebs, she had a huge air cyst in the superior segment. The carbon dioxide laser contracted this air cyst within a minute, and the patient did well. For an extensive bullous lesion, the carbon dioxide laser may be preferable, but for most young patients whose spontaneous pneumothorax is caused by a ruptured bleb, conventional, inexpensive electrocautery is satisfactory.

In conclusion, the thoracoscopic ablation of blebs with electrocautery in treating spontaneous pneumothorax is simple, safe, and effective and warrants further trials.

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Prevalence of Reoperation for Pathway Obstruction After Fontan Operation

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Among 334 patients undergoing the Fontan operation at two institutions and followed for a long time, freedom from reoperation for Fontan pathway obstruction was 99% at 1 year after operation and 96%, 86%, and 59%, respectively, at 5, 10, and 15 years after operation. The hazard function for reoperation had an initial very rapidly declining phase and a second phase that was still rising 15 years after operation. Twelve (17%; confidence limits [CL], 13% to 23%) of 69 patients with a right atrial to right ventricular conduit (valved or nonvalved) connection underwent reoperation for pathway obstruction, whereas only 2 (4%; CL, 1% to 10%) of 45 patients with a direct connection to the right ventricle required reoperation. Five (8%; CL, 4% to 13%) of 65 patients with a right atrial to pulmonary artery conduit connection required reoperation for pathway obstruction, and only 2 (1.3%; CL, 0.4% to 3%) of 155 patients with a direct right atrial to pulmonary artery connection required reoperation. These findings have implications as to the optimal technique of the Fontan operation and also as to use of an intraatrial tunnel as part of the repair.

(Ann Thorac Surg 1989;48:654–9)

The Fontan operation has provided a new and better way of life for many patients born with only one hemodynamically competent ventricle. However, reoperations for obstruction of the surgically created pathways from the systemic venous system to the pulmonary arteries have become more prevalent as the follow-up has lengthened, marring the intermediate and long-term results of the operation. This study seeks a reduction in the incidence of these reoperations by developing information about the incremental risk factors for pathway obstruction leading to reoperation.

Material and Methods

Outcome Event

The outcome event for this study was reoperation for Fontan pathway obstruction during the hospitalization in which the Fontan operation was performed (n = 2) or later (n = 18), either in the institution in which the Fontan operation was performed or in another institution. An additional patient was included in this category in all data, for a total of 21 patients, because she clearly had pathway obstruction confirmed by autopsy but died just before anticipated reoperation.

Patients

All patients undergoing a first Fontan operation at the University of Bordeaux, France (UBF) from 1968 to August 1988 (160 patients) and at The University of Alabama at Birmingham (UAB) from 1975 to March 1988 (174 patients) were included in this study. One-hundred eighty-three patients had tricuspid atresia, 85 had a double-inlet ventricle, 20 had a hypoplastic ventricle, 14 had pulmonary atresia with intact ventricular septum, and 13 had mitral atresia. Nineteen patients had other miscellaneous complex cardiac anomalies. Among these 334 patients, there were 174 male and 160 female patients. Ages ranged from 8 months to 37.5 years (median age, 7 years; mean age ± standard deviation, 9.8 ± 7.3 years). Previous palliative operations had been performed in 265 of the patients.

Surgical Procedure

All patients were operated on through a median sternotomy using extracorporeal circulation with various degrees of hypothermia (20° to 28°C). Pulsatile flow was used since 1978 at UBF and, to avoid excessive hemodilution, ultrafiltration during the latter stages of cardiopulmonary bypass was used in both institutions in the last 2 years. Myocardial protection was achieved with external cardiac cooling and cold crystalloid cardioplegia (since 1976 in UBF) and cold crystalloid or blood cardioplegia (since 1977 at UAB). All previously constructed systemic–pulmonary arterial shunts were taken down, but Glenn anastomoses were left in place. The intracardiac part of the repair was performed according to the anatomy of the malformation, and right atrial to pulmonary artery continuity was established directly or by way of a hypoplastic right ventricle using various techniques.

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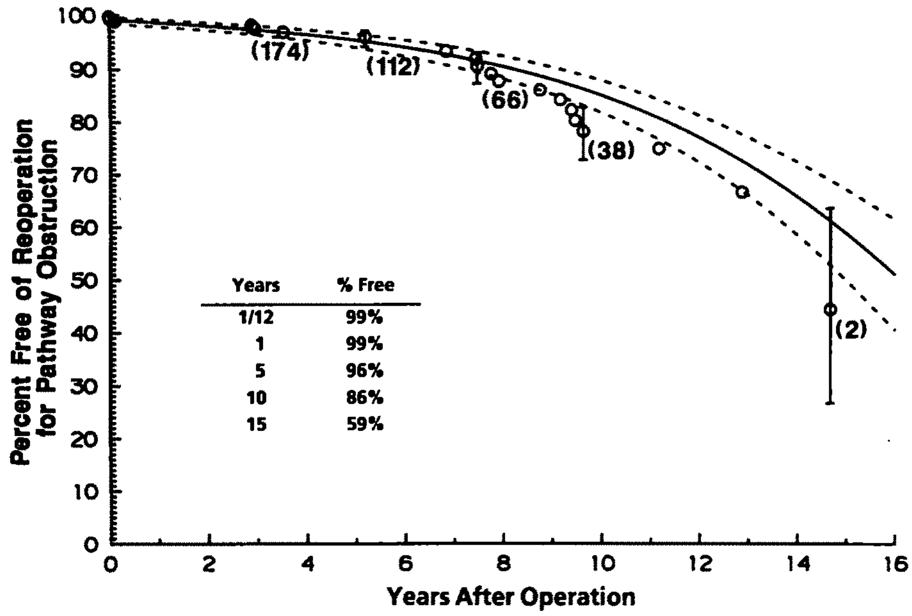


Years	% Free
1/12	99%
1	99%
5	96%
10	86%
15	59%

Fig 1. Time-related freedom from reoperation for pathway obstruction after Fontan operation (UBF, 1968 to August 1988; UAB, 1975 to March 1988; n = 334; 21 reoperations for pathway obstruction). Numbers in parentheses are numbers of patients traced at that interval after operation. Each circle *represents a patient who experienced the event; circles are positioned on the horizontal axis according to the interval between Fontan operation (time zero) and event and are actuarially positioned along the vertical axis.* Vertical bars *represent 70% confidence intervals (±1 standard deviation).* Solid line *represents the parametric estimate of freedom from the event, and* dashed lines enclose *70% confidence intervals. (See Appendix A for parameter estimates for this figure.)*

Method of Study

All documents available in both institutions were reviewed. Particular attention was paid to the details of the construction of the Fontan pathway at the original operation as recorded in the operative notes and the nature of any pathway obstruction that may have developed.

Follow-up Methods

The patients from UBF were followed during September 1988. Nine patients could not be reached at that time. One of these was last seen at his original hospital discharge in 1977. The others among the 8 were last seen in October 1980 (1 year after operation), January 1984 (1 year after operation), January 1984 (5 years after operation), June 1985 (11 years after operation), November 1985 (4 months after operation), March 1986 (8 years after operation), June 1986 (14 years after operation), and October 1986 (6 months after operation), respectively. The median follow-up for the surviving UBF patients was 7.1 years (range, 1 month to 20 years).

The patients from UAB were followed during May 1988. Only 3 patients not known to be dead could not be reached. One of these patients was last seen in October 1987 (6 years after operation), whereas 1 of the other 2 was known to be alive in November 1987 (2 years after operation) and the other was known to be alive in December 1987 (11 years after operation). The median follow-up of surviving patients was 3.9 years (range, 3 months to 12.1 years). The median follow-up for the combined group was 5 years (range, 1 month to 20 years).

Data Base

All information concerning all 334 patients was entered into a single data base; the same criteria were used to describe all events and variables.

Method of Analysis

All reoperations of any type after the Fontan operation were tabulated, including those performed in other institutions and discovered at the time of follow-up inquiry. The reoperations were tabulated according to whether they were performed at the same hospitalization as the Fontan operation or later (Appendix Table A-1). From this list were selected the 21 first reoperations (see section on Outcome Event) considered to have been performed for obstruction of the pathway between the systemic venous system and the pulmonary arteries.

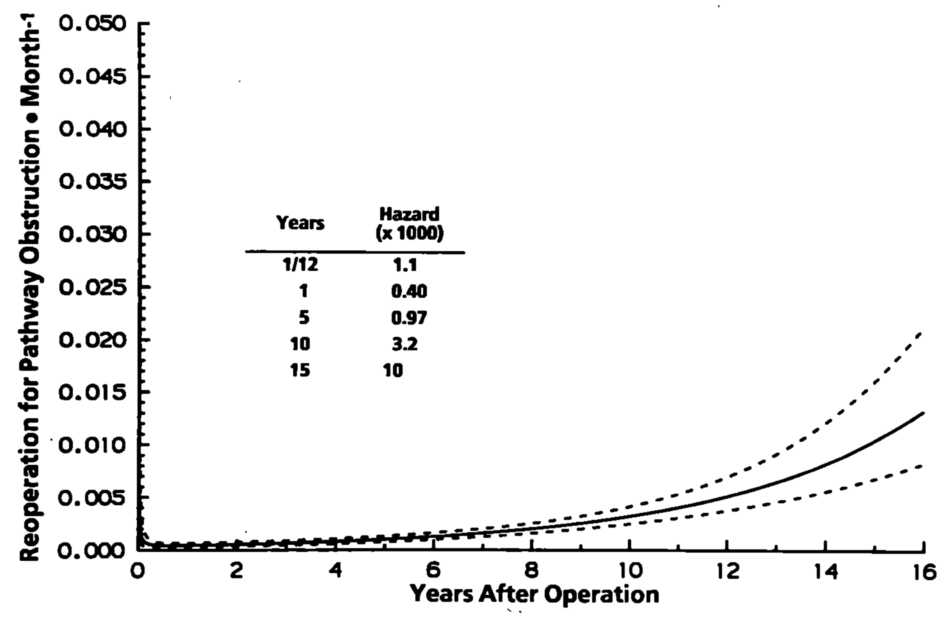
The prevalence of reoperation under varying circumstances was tabulated and was also studied in a time-related manner by actuarial and parametric methods [1, 2].

Results

Freedom From Reoperation

Ninety-nine percent of patients were free from reoperation for pathway obstruction at the end of the first postoperative year, but only 59% were free from such reoperation at the end of 15 years (Fig 1). The hazard function (instantaneous risk) of reoperation was still rising at the end of the follow-up period (Fig 2). A second reoperation was performed in 2 of the 21 patients undergoing a first reoperation, in both of whom the original operation had included a right atrial to right ventricular connection.

Five (24%; CL, 14% to 37%) of the 21 patients undergoing reoperation for Fontan pathway obstruction died in the hospital; the 5-year parametric survival of the group of 21 was 55%.



Years	Hazard (x 1000)
1/12	1.1
1	0.40
5	0.97
10	3.2
15	10

Fig 2. Hazard function (instantaneous risk of event) for reoperation for pathway obstruction after Fontan operation. Data base and equations are the same as for Figure 1, and the depiction is similar. Two phases were present, an early rapidly declining phase and a second slowly rising phase. (See Appendix A for parameter estimates for this figure.)

Risk Factors for Reoperation

The method of construction of the Fontan pathway was clearly an important variable affecting the prevalence of reoperation. Nonvalved direct connections between the right atrium and either the right ventricle or the pulmonary arteries had a low prevalence of reoperations for pathway obstruction (Table 1). In contrast, valved and nonvalved conduit connections had a considerably higher prevalence of reoperations for pathway obstruction, and the 70% confidence intervals for reoperation in this group did not overlap with those for reoperation in patients who had a direct connection, either in right atrial connections to the right ventricle or to the pulmonary artery (Table 1). Conduit connections to the right ventricle, however, had a higher prevalence of reoperations than did those to the pulmonary artery.

With regard to time after first operation, both groups with conduit connections had a steadily declining freedom from reoperation for pathway obstruction, reaching 50% within 15 years (Fig 3), with a corresponding steadily rising hazard function (Fig 4). Although the events were few in both groups with direct connections, patients with a direct connection to the right ventricle had a lesser freedom from reoperation than did patients with a direct connection to the pulmonary arteries, and the difference may not have been a result of chance alone (slightly overlapping 70% confidence intervals).

Multivariate analysis added no new information or insights.

Comment

The analysis indicates that the probability of reoperation for pathway obstruction is slight when no conduit, valved or unvalved, is in the right atrial to right ventricular or pulmonary arterial pathway. With a conduit in the pathway, however, the inevitability of reoperation is suggested. When the conduit is between the right atrium and right ventricle, sternal compression of the conduit may increase the probable need for reoperation.

A previous study of survival and functional status after the Fontan operation in patients in whom reoperation for pathway obstruction was not needed identified a right atrial to pulmonary artery connection (rather than one to

Table 1. Type of Distal Right Atrial Connection in Fontan Operation and Prevalence of Reoperation for Pathway Obstruction

Site and Type of Distal Right Atrial Connection	n	Reoperation for Pathway Obstruction No.	%	70% Confidence Limits (%)
Right ventricle	114	14	12	9–16
Conduit	69	12	17	13–23
Valved	20	2	10	3–22
Heterograft	1	0	0	0–85
Homograft	19	2	11	4–23
Nonvalved	49	10	20	14–28
Direct	45	2	4	1–10
Simple	32	2	6	2–14
Roofed	13	0	0	0–14
Pulmonary artery	220	7	3	2–5
Conduit	65	5	8	4–13
Valved	63	5	8	4–13
Heterograft	11	1	9	1–28
Homograft	52	4	8	4–14
Nonvalved	2	0	0	0–61
Direct	155	2	1.3	0.4–3
Simple	118	2	2	0.6–4
Roofed	37	0	0	0–5
Total	334	21	6	5–8

These operations (n = 334) were performed at the University of Bordeaux, France, between 1968 and August 1988 and at The University of Alabama at Birmingham between 1975 and March 1988.

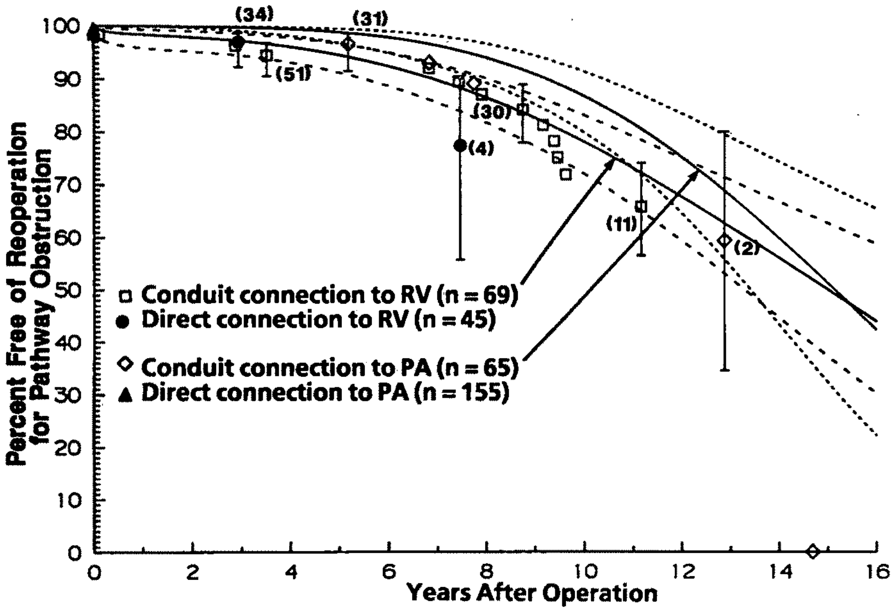


Fig 3. Stratified depiction of freedom from reoperation for pathway obstruction according to distal right atrial connection. Symbols are positioned along the horizontal axis at the time of event and are positioned actuarially along the vertical axis. Vertical bars *represent actuarial 70% confidence intervals (±1 standard deviation).* Solid lines, *with their 70% confidence intervals enclosed by* dashed lines, *represent parametric estimates for the two groups with conduit connections. (See Appendixes B and C for parameter estimates for this figure.)*

the right ventricle) as a risk factor for those events [3]. That study was not able to identify an advantage in this regard of valved or nonvalved conduits or direct connections to the right ventricle. However, in view of the morbidity and hospital mortality (24%) or reoperations for pathway obstruction evident in the present study, and the seemingly nearly inevitable need for reoperation when conduits are used in the connection, direct (rather than conduit) connections appear to be indicated for right atrial connections to either the ventricle or pulmonary artery as part of the Fontan operation.

The study does not allow a secure inference as to the possibility that, in the absence of sternal compression, a homograft valved conduit between right atrium and right ventricle would require fewer reoperations than Dacron conduits. This is unfortunate, because from other viewpoints [4, 5] a homograft valved connection between right atrium and right ventricle appears to be optimal for patients with tricuspid atresia and concordant ventriculoarterial connections who are aged 10 years or older and thus beyond their important growth phase. Perhaps this could be constructed as a planned second stage after an initial direct right atrial to right ventricular connection performed earlier in life.

The results of the study suggest the possibility that obstruction may also develop in synthetic tunnels within the right atrium to connect the inferior vena cava to the superior vena cava as a part of a Fontan operation. Whether incorporation of right atrial wall as a part of the tunnel circumference will prevent this [6–8] remains to be seen, but the data encourage making as much as possible of the circumference of such tunnels from right atrial wall.

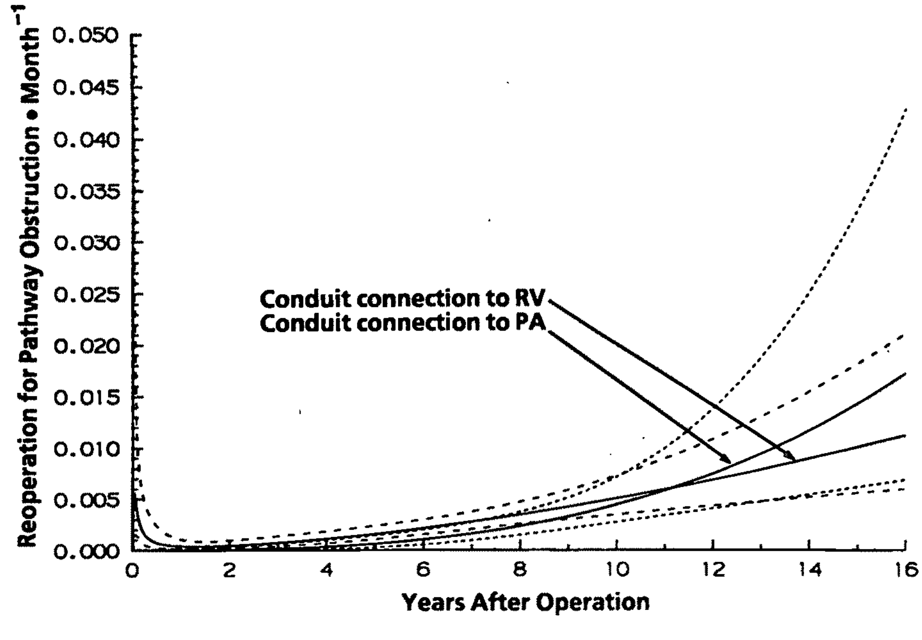


Fig 4. Hazard function for the two conduit groups. Data base and equations are the same as for Figure 3, and the depiction is similar. (See Appendixes B and C for parameter estimates for this figure.)

We appreciate the willingness of Drs A. D. Pacifico and J. K. Kirklin at UAB to have patients operated on by them included in the study and also appreciate their helpful comments and criticisms during preparation of the manuscript. We also acknowledge the help and intimate cooperation in management of these patients given by Dr A. Choussat (UBF) and Drs Bargeron, Colvin, and McConnell (UAB). We gratefully acknowledge the assistance in data collection and analysis of Brooks Counts, Germain LeGoff, and Christian Nuillemin, and the assistance of Debbie Nuby in preparation of the tables, graphics, and manuscript.

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Appendix Table A1. All Reoperations Early and Late After Fontan Operation (UBF 1968 to August 1988; UAB 1975 to March 1988; n = 334)

		Hospital Deaths			Total Deaths	
Type of Reoperation	n	No.	%	70% CL (%)	No.	%
Early reoperations						
Reoperation for pathway obstruction (initially RA–PA direct connection)	2	1	50	7–93	1	50
Takedown of Fontan operation	4	2	50	18–82	2	50
Glenn anastomosis	4	3	75	37–97	4	100
Repair of AV valve patch leak	4	1	25	3–63	2	50
Re-repair of ASD	3	1	33	4–76	1	33
Operations for fluid removal	1	1	100	15–100	1	100
Miscellaneous	5	0	0	0–32	1	20
Subtotal	23	9	39	27–52	12	52
Late reoperations						
Reoperation for pathway obstruction	19[a]	4	21	11–35	6	32
Initially RA–RV conduit, allograft valved	2	0	0	0–61	0	0
Initially RA–RV conduit, nonvalved	10	1	10	1–30	2	20
Initially RA–RV direct	2	1	50	7–93	1	50
Initially RA–PA conduit, allograft valved	4	2	50	18–82	3	75
Initially RA–PA conduit, heterograft valved	1	0	0	0–85	0	0
Re-repair of ASD	5	1	20	3–53	1	20
Takedown of Fontan operation	3	1	33	4–76	1	33
Glenn anastomosis	4	2	50	18–82	3	75
Operations for fluid removal	7	2	29	10–55	2	29
Enlargement of VSD	3	0	0	0–47	0	0
Repair of AV valve patch leak	3	1	33	4–76	1	33
Cardiac transplantation	4	2	50	18–82	2	50
Miscellaneous	5	1	20	3–53	2	40
Subtotal	53	14	26	20–34	18	34
Total	76	23	30	24–37	30	39

These operations (n = 334) were performed at the University of Bordeaux, France, between 1968 and August 1988 and at The University of Alabama at Birmingham between 1975 and March 1988. Early reoperations were performed during the hospitalization in which the Fontan operation was performed; late reoperations were performed subsequent to that hospitalization, and some were performed in other institutions.

[a] One of these 19 patients died before reoperation (see Material and Methods section).

ASD = atrial septal defect; AV = atrioventricular; CL = confidence limits; PA = pulmonary artery; RA = right atrium; RV = right ventricle; VSD = ventricular septal defect.

Appendix Table A2. Relation Between Cardiac Morphology and the Type of Distal Right Atrial Connection and the Prevalence of Reoperation for Pathway Obstruction

Morphologic Category and Site of Distal Right Atrial Connection	n	Reoperation for Pathway Obstruction		
		No.	%	70% CL (%)
Tricuspid atresia	183	19	10	8–13
RV connection	104	14	13	10–18
Conduit	63	12	19	14–26
Valved (homograft)	14	2	14	5–31
Nonvalved	49	10	20	14–28
Direct	41	2	5	2–11
Simple	28	2	7	2–16
Roofed	13	0	0	0–14
PA connection	79	5	6	4–11
Valved conduit	29	4	14	7–24
Heterograft	3	1	33	4–76
Homograft	26	3	12	5–22
Direct	50	1	2	0.3–7
Simple	40	1	2	0.3–8
Roofed	10	0	0	0–17
Other morphology	151	2	1	0.4–3
RV connection	10	0	0	0–17
Valved conduit	6	0	0	0–27
Heterograft	1	0	0	0–85
Homograft	5	0	0	0–32
Direct, simple	4	0	0	0–38
PA connection	141	2	1	0.5–3
Conduit	36	1	3	0.4–9
Valved (heterograft)	8	0	3	0.4–10
Valved (homograft)	26	1	4	0.5–12
Nonvalved	2	0	0	0–61
Direct	105	1	1	0.01–3
Simple	78	1	1	0.2–4
Roofed	27	0	0	0–7
Total	334	21	6	5–8

These operations (n = 334) were performed at the University of Bordeaux, France, between 1968 and August 1988 and at The University of Alabama at Birmingham between 1975 and March 1988; there were 21 reoperations for pathway obstruction. By χ^2 test, $p = 0.12$ for RV connection versus PR connection in tricuspid atresia.

CL = confidence limits; PA = pulmonary artery; RV = right ventricle.

Appendix A

For the time-related freedom from reoperation for pathway obstruction, the shaping parameters for the parametric estimates (Figs 1 and 2) were as follows. Early phase: $\delta = 0$, $\rho = 0.07198$, $\nu = 2.906$, $m = 0$, intercept $= 0.01213$; Late phase: $\tau = 1$, $\gamma = 1$, $\alpha = 1$, $\eta = 2.827$, intercept $= 2.233 \times 10^{-7}$.

Appendix B

For reoperation for Fontan pathway obstruction in patients receiving a right atrial to right ventricular conduit connection (n = 69), the parameter estimates are as follows. Early phase: $\delta = 0$, $\rho = 2.270$, $\nu = 1.701$, $m = 0$, $\mu_1 = 0.02228$; Late phase: $\tau = 1$, $\gamma = 1$, $\alpha = 1$, $\eta = 2.717$, $\mu_3 = 4.988 \times 10^{-7}$.

Appendix C

For reoperation for Fontan pathway obstruction in patients receiving a right atrial to pulmonary arterial conduit connection (n = 65), the parameter estimates are as follows. Late phase: $\tau = 1$, $\gamma = 1$, $\alpha = 1$, $\eta = 3.884$, $\mu_3 = 1.156 \times 10^{-9}$.

Reconstruction of the Esophagus With the Left Colon

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This report reviews our experience with 96 patients with benign or malignant stricture of the esophagus who underwent interposition of the left colon with or without esophageal resection from July 1982 to June 1987. There were 67 male and 29 female patients ranging in age from 8 to 80 years. Thirty-seven patients had fibrotic stricture secondary to corrosive injury of the esophagus, 42 had cancer of the esophagus, and 17 had cancer of the gastric cardia. The incidence of postoperative complications and surgical mortality, respectively, was 16.2% and 2.7% for patients with corrosive stricture of the esophagus, 35.7% and 11.9% for patients with cancer of the esophagus, and 35.2% and 5.8% for patients with cancer of the gastric cardia. Reconstruction resulted in good function in 75.6% of the patients with corrosive stricture of the esophagus, 66.6% of the patients with cancer of the esophagus, and 70.5% of patients with cancer of the gastric cardia. The morbidity and mortality were higher in the group with malignant esophageal strictures because of advanced age, poor general condition of the patient, and extent of the surgical procedure needed. Cervical anastomotic leakage was the most frequently encountered complication (13.5%), and all the poor-function results were caused by this complication. In our experience, reconstruction of the esophagus with left colon is a satisfactory method that can be accomplished with acceptable morbidity and mortality. The left colon is a durable and functional substitute.

(Ann Thorac Surg 1989;48:660–4)

Restoration of the continuity of the alimentary tract and the resumption of the normal swallowing mechanism poses a challenging problem in the management of benign and malignant stricture of the esophagus. The use of the left colon, either for replacement or bypass of the esophagus, was reported in 1911. Kelling [1] used the transverse colon in the isoperistaltic position as an esophageal substitute, and Vulliet [2] described use of the transverse colon but placed it in an antiperistaltic position. Since then, the left colon has become a favored organ for reconstruction of the esophagus because it is easy to isolate and has an excellent blood supply from the concomitant vessels with the marginal artery of Drummond.

We have used the left colon as an esophageal substitute in 96 patients, and describe here the operative techniques and the results. We conclude that using the left colon as an esophageal substitute can be accomplished with acceptable morbidity and mortality.

Material and Methods

This series consists of 96 patients in whom reconstruction of the esophagus was accomplished with the left colon during the 5-year period July 1982 to June 1987. Among the 96 patients, 37 underwent esophageal reconstruction for corrosive stricture of the esophagus. Their age ranged from 8 to 62 years. Forty-two patients with cancer of the esophagus underwent esophageal reconstruction (22 palliative bypass procedures and 20 resections with reconstruction). They ranged in age from 49 to 80 years. Seventeen patients with cancer of the gastric cardia underwent total gastrectomy, distal esophagectomy, and reconstruction of the esophagus with intrathoracic interposition of a segment of colon. Their age ranged from 56 to 70 years (Table 1).

Preoperative Preparation

Bowel preparation consisted of a low-residue diet and warm saline solution enemas for two days, then a clear liquid diet with oral administration of 1 g of neomycin or erythromycin for four doses, and finally a cleansing enema on the day before operation. If there were no symptoms or signs of colonic disease, barium enema or colonoscopy was not routinely performed.

Operative Technique for Corrosive Stricture

The operation is carried out by the two-team approach. The patient is put in the supine position with the head rotated to the right and the neck hyperextended for proper exposure of the cervical region. Standard endotracheal general anesthesia is used. The skin is prepared from the tip of the left mastoid process to the pubis.

The operation starts with an abdominal exploration through a long midline incision. Exploration is focused particularly on the colon to determine its suitability and on the stomach to determine if there is any intrinsic disease or damage caused by the caustic agent. After

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Table 1. Summary of Preoperative Patient Data

Diagnosis	No. of Patients	Age (yr)	Sex (M/F)
Corrosive stricture	37	33 (8–62)	14/23
Carcinoma of esophagus	42	62 (49–80)	37/5
Carcinoma of cardia	17	62 (56–70)	16/1
Total	96	. . .	67/29

proper evaluation, preparation of the left colon based on the left colic artery for isoperistaltic transplantation is carried out.

The colon is mobilized by incising the lateral peritoneal reflection from the sigmoid to the splenic flexure and by incising the relatively avascular attachments of the greater omentum to the transverse colon. The lienocolic attachments to the splenic flexure are divided. The descending colon, splenic flexure, and transverse colon can now be held up.

The mesocolon is transilluminated and its vascular pattern is examined (Fig 1). The flow in the marginal artery is examined by palpation. The site of division of the transverse colon and its marginal artery and the descending colon and its marginal artery are selected in relation to the length of substitute needed (Fig 2). Nothing is divided until these vessels have been temporarily obstructed with bulldog clamps at the sites of their planned division and show adequate terminal perfusion (Fig 3). A long segment of colon is obtained by dividing the middle colic artery proximal to its major bifurcation and by dividing the colon distal to both branches of the left colic artery (Fig 4).

Meanwhile, the second team explores the cervical esophagus through an oblique incision in the left side of the neck anterior to the sternocleidomastoid muscle. The prevertebral plane is entered by retracting the carotid sheath laterally. The esophagus is exposed and encircled with tape; care is taken not to injure the recurrent laryngeal nerve. The esophagus is transected as low as possible and the distal end is closed with sutures. An extrapleural retrosternal tunnel is constructed by blunt finger dissection from both ends of the sternum. The prepared colonic segment and its vascular pedicle are then brought up through this tunnel in an isoperistaltic manner to the neck where it should lie comfortably without tension. Special care is taken not to twist the colonic segment because this could compromise the venous return.

The two teams then carry out the three necessary anastomoses. The performance of the esophagocolic anastomosis is the most critical and must be done meticulously. The cologastric anastomosis is carried out end-to-side to the anterior aspect of the midbody of the stomach or to the jejunum if the stomach has been damaged by the caustic agent. Pyloroplasty is performed as needed but not routinely. A chest roentgenogram is made immediately after operation to check whether pneumothorax is present.

Operative Technique for Cancer of Esophagus

Five of the 42 patients in this group had tracheoesophageal fistula, poor pulmonary function, or distant metas-

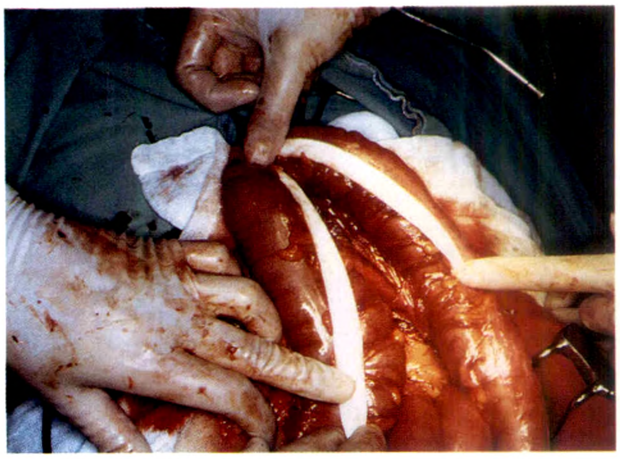

Fig 2. The length of the substitute is measured so that the transplant can be brought to the neck without tension.

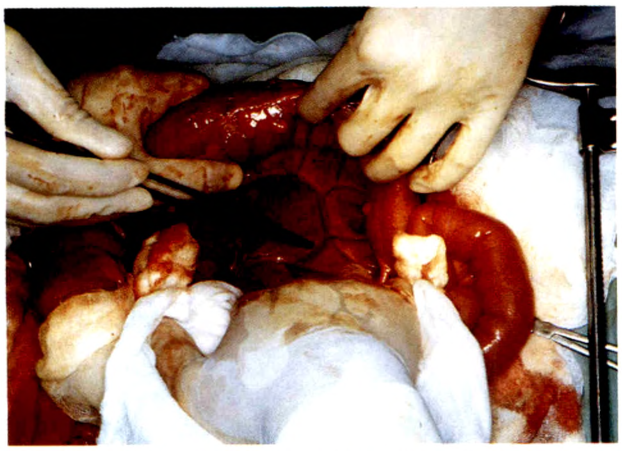

Fig 1. After the transverse colon and descending colon have been mobilized, the vascular pattern is examined and the left colic artery is visible.

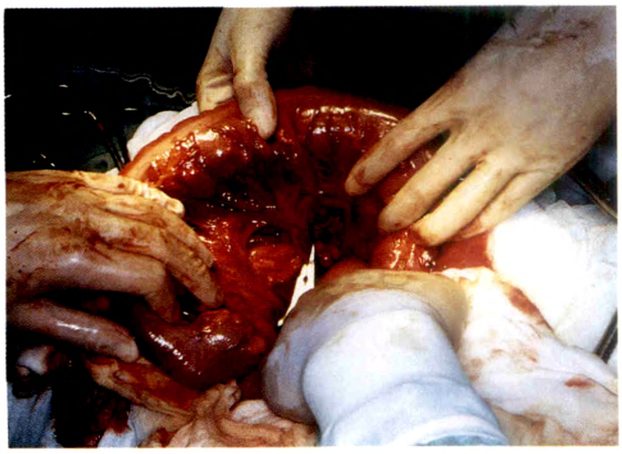

Fig 3. The middle colic artery and vein are obstructed with bulldog clamps to make sure that perfusion of the transplant is adequate.

tasis, and received only palliative colon bypass with the technique just described. The other 37 patients underwent initial exploratory thoracotomy. Only 28 were found to have an unresectable lesion and underwent palliative colon bypass.

The other 9 patients had resection of the esophagus through a right posterolateral thoracotomy with repositioning of the patient in the supine position for reconstruction. Because the stomach in these patients had intrinsic disease or had been resected in the past, the left colon was used for reconstruction. (Normally, the stomach is our choice for replacement of the esophagus.)

Operative Technique for Cancer of Cardia of Stomach

Seventeen patients underwent total gastrectomy and distal esophagectomy through a left thoracoabdominal incision with reconstruction of the esophagus. An intrathoracic short-segment colon interposition was anastomosed distally to the jejunum.

Evaluation of Functional Results

The patient who tolerates a solid or soft diet without complaint is classified as having "good" function. If mild dysfunction is present but the patient can tolerate a soft or semiliquid diet with the help of intermittent dilation and maintains satisfactory nutrition, he or she is classified as having "fair" function. When major dysphagia persists and the patient must have regular dilation or revision o the anastomosis, he or she is classified as having "poor function.

Table 2. Complications[a]

Complication	Corrosive Injury (n = 37)	Cancer of Esophagus (n = 42)	Cance of Cardi (n = 1
Anastomotic leakage	3	8	2
Bile reflux	0	0	2
Wound infection	0	2	1
Suffocation	0	1	0
Necrosis of transplant	1	0	0
Vocal cord palsy	1	2	0
Intestinal obstruction	1	0	0
Pneumothorax	0	0	1
Empyema	0	1	0
Wound disruption	0	1	0
Total	6 (16.2)	15 (35.7)	6 (35.2

[a] Numbers in parentheses are percentages.

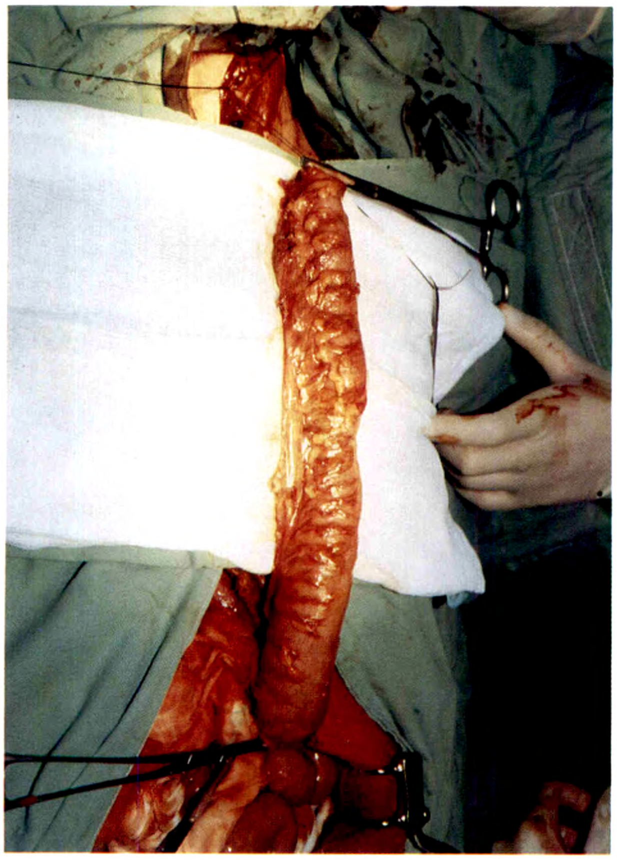

Fig 4. The substitute is taken with adequate length and good perfusion such that the reconstruction can be performed without tension.

Results

For the 37 patients having reconstruction for corrosiv stricture of the esophagus, the overall complication rat was 16.2% (6/37) with three cervical anastomotic leaks one necrosis of the transplant, one vocal cord palsy, an one intestinal obstruction. There was only one death, fo a mortality rate of 2.7% (1/37). The patient with necrosis o the transplant died of sepsis on the tenth postoperativ day. The functional result was good in 75.6% of th patients, fair in 16.2%, and poor in 8.1%.

In the group with cancer of the esophagus, the overal complication rate was 35.7% (15/42) with eight cervica anastomotic leaks, two wound infections, two vocal cor palsies, one suffocation, one empyema, and one woun disruption. The overall mortality rate was 11.9% (5/42) Two patients died of pneumonia, 2 died of sepsis, and died of suffocation. The functional outcome was as fol lows: good, 66.6% of the patients; fair, 21.4%; and poor 11.9%.

For the group with cancer of the gastric cardia, th overall complication rate was 35.2% (6/17) with two anas tomotic leaks at the intrathoracic esophagocolostomy, tw cases of bile reflux, one wound infection, and one pneu mothorax. There was one death, which was due to sepsis for a mortality rate of 5.8% (1/17). The functional result were as follows: good, 70.5% of the patients; fair, 17.6% poor, 11.7% (Tables 2–4).

Comment

When reconstruction of the esophagus is indicated in patient with dysphagia caused by either malignant o benign stricture of the esophagus, there are several sub stitutes from which to choose. An ideal substitute mus

Table 3. Causes of Death[a]

Cause	Corrosive Injury (n = 37)	Cancer of Esophagus (n = 42)	Cancer of Cardia (n = 17)
Pneumonia	0	2	0
Sepsis	1	2	1
Suffocation	0	1	0
Total	1 (2.7)	5 (11.9)	1 (5.8)

[a] Numbers in parentheses are percentages.

fulfill several criteria, including complete and permanent relief of dysphagia, no late complications or sequelae, acceptable operative mortality and morbidity rates, ability to replace the entire esophagus and hypopharynx where necessary, technique applicable to infants and children, and technique allowing resection and reconstruction in a single-stage procedure. In addition, it is helpful if the technique can be taught to the resident or trainee of average ability [3].

If there is no substitute that fulfills all of these criteria, the surgeon must individualize the choice. The stomach is the organ most frequently used for substitution or bypass of a malignant stricture of the esophagus unless it has intrinsic disease such as peptic ulcer, has been operated on previously, or is to be included in the resection. The stomach provides adequate length and excellent blood supply from the right gastric and right gastroepiploic arteries, and requires only one anastomosis. The major disadvantage of using the stomach as an esophageal substitute is the frequency of reflux. Skinner and Belsey [4] reported a 17% incidence of severe esophagitis after reconstruction of the esophagus with stomach for benign stricture, and suggested reconstruction with colon for patients with an anticipated long life expectancy.

The advantages of interposition of an isoperistaltic segment of jejunum for esophageal reconstruction are its active peristalsis and rarity of subsequent intrinsic jejunal disease. The disadvantage is that the "bowstring" vascular pedicle is accompanied by a long loop of redundant jejunum. Because of this disadvantage, the jejunum is seldom used for reconstruction.

Despite the necessity for three anastomoses, the colon is a very satisfactory substitute for the esophagus. The advantages of the left colon include the following: the isoperistaltic transplant is of sufficient length to replace the entire esophagus; the blood supply from the left colic artery is robust and rarely prone to anastomotic variation; and no redundancy or kinking, frequently seen with jejunum, is encountered. However, there are several contraindications to its use: variation in the vascular pattern of the marginal arteries; marked mesenteric arteriosclerosis; and intrinsic colon disease such as diverticulosis, diverticulitis, polyps, or carcinoma.

Table 4. Function of Substitute[a]

Patient Group	Good	Fair	Poor
Corrosive injury	28 (75.6)	6 (16.2)	3 (8.1)
Cancer of esophagus	28 (66.6)	9 (21.4)	5 (11.9)
Cancer of cardia	12 (70.5)	3 (17.6)	2 (11.7)
Total	68 (70.8)	18 (18.7)	10 (10.4)

[a] Numbers in parentheses are percentages.

Preference for the left colon over the right results from its more consistent marginal vessels. The marginal artery of the right colon may be incomplete in as many as 70% of patients. The lack of continuity is most often between the ileocolic and right colic arteries or between the middle and right colic arteries near the hepatic flexure. In 12.6% of patients, the right colic artery was absent altogether. In addition, the right colon can have relatively poor venous drainage [5–7]. Wilkins [8] reported that necrosis developed in 75.6% of 32 right colon replacements, but in only 2.9% of 68 left colon substitutions. Preoperative inferior mesenteric angiography was used in the patients seen early in our series to evaluate the vascular pattern of the left colon; we found no defect in the marginal artery in those patients. This invasive examination was then abandoned and the vascular condition was evaluated directly at operation. Only 3 patients were found to have a questionable blood supply, and in them, jejunum was used as the substitute. Although there are three anastomoses in colon procedures, the anastomoses are carried out by two teams of surgeons to shorten the operative time.

Among our 96 patients who had reconstruction, the morbidity rate was much higher for those with malignant disease (35.7%) of the esophagus than benign stricture (16.2%). We have found several factors that might contribute to the difference. (1) The mean age of patients with benign stricture of the esophagus (32.9 years) was much younger than that of those with malignant stricture (61.6 years). (2) In the group with corrosive injury of the esophagus, the operation is not as extensive as for malignant stricture because the patient requires only a laparotomy and cervical incision for the bypass operation; but with malignant stricture of the esophagus, the patient must undergo thoracotomy for resection of the esophagus or exploration before palliative bypass, thus resulting in some pulmonary complications. (3) The general condition of the patient with malignant stricture is poorer than that of the patient with benign stricture, especially in the late stages. For the same reasons, the mortality rate is also much lower (2.7%) for patients with benign stricture than those with malignant stricture (11.9%, 5.8%).

Cervical anastomotic leakage is still the leading complication encountered in esophageal reconstruction. The incidence was only 8.1% for patients with corrosive injury of the esophagus, but 19% for patients with cancer of the esophagus and 11.7% for those with cancer of the gastric cardia. The most important factors in preventing this complication are preservation of the marginal artery, gentle handling of the transplant, maintenance of venous return, and creation of a roomy tunnel [9].

The overall morbidity and mortality rates in this series are similar to those in a previous series of our patients having reconstruction of the esophagus with stomach (morbidity, 31.8%, and mortality, 4.5%) [10].

As for the function of the substitute, 70.8% of patients undergoing reconstruction with colon regained good swallowing function, 18.7% obtained fair function, and only 10.4% had poor function. Most of the poor-function results were due to cervical anastomotic leakage. Unusual attention to details of technique is mandatory to avoid complications and late swallowing problems.

We thank Dr Earle W. Wilkins, Jr, for reviewing the manuscript.

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Amrinone Versus Conventional Therapy in Pulmonary Hypertensive Patients Awaiting Cardiac Transplantation

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Pulmonary hypertension is associated with an increased perioperative mortality for orthotopic heart transplantation. A transpulmonary gradient greater than 15 mm Hg or a pulmonary vascular resistance greater than 5 Woods units increases mortality secondary to right heart failure. This study compares amrinone with conventional therapy in 38 transplant candidates with pulmonary hypertension. All patients had elevated transpulmonary gradient, pulmonary vascular resistance, or both. Group 1 (n = 21) received prolonged continuous intravenous amrinone therapy, whereas group 2 (n = 16) received high-dose oral diuretics, digitalis, and captopril. Both groups 1 and 2 had decreased pulmonary hypertension, transpulmonary gradient, and pulmonary vascular resistance. However, amrinone was more effective, with a 86% response rate versus 63% response for conventional therapy. Survival awaiting transplantation was significantly higher in group 1 (20 of 22, 91%) than in group 2 (10 of 16, 63%). Although both groups 1 and 2 had significantly decreased pulmonary vascular resistance, only group 2 had significantly decreased systemic vascular resistance. Comparison of pulmonary vascular resistance after therapy showed that the response in group 1 (amrinone) was significantly lower than the response in group 2 (conventional therapy), suggesting that amrinone may function as a direct vasodilator of the pulmonary vasculature. There were no operative deaths or episodes of perioperative right heart failure in either group. Amrinone appears to be more effective and safe than conventional therapy in the treatment of prospective heart transplant candidates with pulmonary hypertension.

(Ann Thorac Surg 1989;48:665–9)

Pulmonary hypertension (PHT) and elevated pulmonary vascular resistance (PVR) increase perioperative mortality for orthotopic heart transplantation. Kormos and associates [1] noted that a transpulmonary gradient (TPG) greater than 15 mm Hg or a PVR greater than 5 Woods units, or both, were associated with an operative mortality of 25% secondary to acute right heart failure. Kirklin and associates [2], using analysis of variance for risk factors in 61 orthotopic heart transplant patients, showed that elevated PVR causes a significant increase in perioperative mortality. Amrinone, a phosphodiesterase inhibitor, was shown by Bolling and colleagues [3] to lower TPG to less than 15 mm Hg and PVR to less than 5 Woods units in 89% of patients when administered with a prolonged intravenous course. The perioperative mortality in this group of patients was 5% [3]. Due to the shortage of suitable donor organs and an average wait of approximately 8 months until transplantation, prolonged intravenous therapy in the hospital setting is not feasible, and more conventional modes of therapy with oral medication in an outpatient setting may be more appropriate.

This article is a retrospective review comparing use of conventional therapy (digitalis, peripheral vasodilators, and high-dose diuretics) in an outpatient setting versus inpatient continuous intravenous amrinone in 38 pulmonary hypertensive patients awaiting cardiac transplantation.

Material and Methods

Table 1 is a patient profile comparing the demographics of the two groups. There were no significant differences in age, sex, or cause of disease between the two groups. All patients for transplantation were initially evaluated with right heart catheterization. Patients with PHT, defined as either a TPG greater than 15 mm Hg or PVR greater than 5 Woods units, were administered oxygen and given a trial of intravenous nitroprusside therapy. Those demonstrating an adequate decrease in left ventricular afterload reduction with a subsequent decrease in systemic systolic arterial pressure to less than 90 mm Hg but not showing a significant decrease in pulmonary arterial pressure, TPG, and PVR were admitted to the intensive care unit for more intensive treatment. These select patients were believed to have PHT not immediately responsive to left ventricular unloading.

Thirty-eight patients who were referred for cardiac transplantation demonstrated PHT independent of immediate left ventricular unloading. Group 1 consisted of 22 patients who were treated as inpatients with prolonged intravenous amrinone therapy. Group 2 consisted of 16

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Table 1. Patient Profile

Variable	Group 1	p-Value	Group 2
No. of patients	22	NS	16
Age (yr)[a]	51 (59–37)		53 (62–41)
Sex	20 M, 2 F		14 M, 2 F
Cause of disease			
Ischemic	14 (67%)		13 (81%)
Cardiomyopathy	7 (33%)		3 (19%)
Responders	19 (86%)	0.053	10 (63%)
Days until response[a]	3.5 (0.5–7)	<0.05	8.2 (2–14)
Treatment duration (days)[a]	37 (3–136) days		8 (1.5–18) mo
Survival	20 (91%)	<0.05	10 (63%)
Percentage of responders transplanted	16/19 (84%)		7/10 (70%)
Deaths			
Responders	2 (10%)		3 (33%)
Nonresponders	0		3 (50%)

[a] Numbers in parentheses are ranges.

patients who received digitalis, high-dose diuretics, and an angiotensin-converting enzyme inhibitor (as an unloading agent) in the hospital for 48 hours and were then discharged and followed as outpatients. All patients had clinically significant congestive heart failure (New York Heart Association class III to IV) with primary myocardial disease and no intracardiac shunts.

All patients were initially admitted to the intensive care unit, and a Swan-Ganz catheter and peripheral arterial line were inserted. Baseline hemodynamic values were obtained, including systemic arterial and venous pressure, pulmonary arterial pressure, pulmonary capillary wedge pressure, cardiac output (CO), transpulmonary gradient, systemic and pulmonary vascular resistances, and daily weights. Measurement of serum electrolytes, blood urea nitrogen, and creatinine, tests of liver function, and platelet counts were performed daily for the first three days and then on a weekly basis.

Group 1 patients were administered amrinone in a 0.75-mg/kg loading dose and were then started on a continuous infusion of 2.5 μg/kg/min. Hemodynamic variables were measured hourly for four hours and then every four hours for 72 hours. Infusion was titrated to an upper dose of 20 μg/kg/min to obtain a desired target effect of a TPG of less than 15 mm Hg, a PVR less than 3 Woods units, or both. After 48 hours the monitoring lines were removed and were reinserted only weekly for 1 month and then monthly if a patient became hemodynamically unstable. All patients were continued on intravenous amrinone until transplantation, hemodynamic decompensation, or exclusion from transplantation for other reasons. If a patient became hemodynamically unstable and required additional inotropic or mechanical support, all subsequent pulmonary pressures were excluded from analysis in this study, but the final outcome of the patient was considered in the statistical assessment.

Group 2 patients ($n = 16$) were also admitted to the intensive care unit and underwent the same monitoring. All received digoxin (0.25 mg/day) and diuretics titrated to weight, levels of blood urea nitrogen and creatinine, and fluid balance. Patients also received an oral angiotensin-converting enzyme inhibitor (captopril) until a systemic blood pressure of approximately 90 mm Hg was achieved or symptoms of decreased peripheral perfusion occurred. Hemodynamic variables were also measured hourly for four hours and then every four hours for 72 hours, at which time the monitoring lines were removed and the patients were discharged and catheterized on a weekly basis for 1 month and then monthly; patients were readmitted if they became hemodynamically unstable. Blood studies were monitored on a weekly basis.

Table 2 compares the drug regimens of the two groups. Patients in group 1 received intravenous amrinone at an average dose of 9 μg/kg/min. Nine of the patients were taking diuretics, and the mean furosemide dose was 40 mg/day. Two patients received 5 mg/day of metolazone in addition to furosemide. All 16 patients in group 2 received furosemide at a mean dose of 360 mg/day. In addition, 9 patients received metolazone (10 mg/day) and 5 received bumetanide (6 mg/day). All 16 patients in group 2 received digoxin (0.25 mg/day), and 11 received the angiotensin-converting enzyme captopril at an average dose of 37.5 mg/day. The duration of treatment for group 1 ranged from three to 136 days (mean duration, 29 days). Treatment duration for group 2 was 1.5 to 18 months (mean duration, 8 months).

Results

Table 1 displays the patients' response to therapy. Nineteen of the 22 patients (86%) in group 1 responded to therapy with a TPG of less than 15 mm Hg or a PVR of less than 3 Woods units, or both, whereas 10 of 16 patients (63%) in group 2 responded ($p = 0.053$). Group 1 response time was significantly less than that of group 2 (3.5 days versus 8.2 days; $p < 0.05$). The pretransplantation

Table 2. Drug Regimens of Patients With Pulmonary Hypertension

Group 1 (Amrinone Therapy)			Group 2 (Conventional Therapy)	
Drug	Dose	No. of Patients	Dose (mg/ day)	No. of Patients
Lasix	40 mg/day	9	360	16
Digoxin	. . .	. . .	0.025	16
Captopril	. . .	. . .	37.5	11
Metolazone	5 mg/day	2	10	9
Bumetanide	. . .	. . .	6	5
Amrinone	0.75 mg/kg loading, 9 μg/kg/min	22	. . .	. . .

Table 3. Amrinone Versus Conventional Therapy: Pretherapy Hemodynamic Values

Hemodynamic Variable	Conventional Therapy (n = 16)	*p* Value	Amrinone Therapy (n = 22)
BUN (mg/dL)	27.7 ± 3.5	NS	28.8 ± 2.2
CREAT (mg/dL)	1.4 ± 0.1	NS	1.35 ± 0.06
PAM (mm Hg)	46.2 ± 1.8	NS	44.3 ± 1.5
PCWP (mm Hg)	27.7 ± 1.8	NS	25.2 ± 1.0
TPG (mm Hg)	18.1 ± 1.0	NS	19.0 ± 1.2
CO (L/min)	3.6 ± 0.2	NS	3.5 ± 0.3
PVR (WU)	5.1 ± 0.4	NS	5.8 ± 0.3
MAP (mm Hg)	85 ± 3.6	NS	79.0 ± 3.4
CVP (mm Hg)	11.7 ± 2.0	NS	12.4 ± 2.0
SVR (WU)	21.2 ± 1.3	NS	19.0 ± 2.2

BUN = blood urea nitrogen; CO = cardiac output; CREAT = creatinine; CVP = central venous pressure; MAP = mean arterial pressure; NS = not significant; PAM = mean pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; TPG = transpulmonary gradient; WU = Woods units.

Table 5. Conventional Therapy (n = 16)

Hemodynamic Variable	Pretherapy	*p* Value	Posttherapy
BUN (mg/dL)	27.7 ± 3.5	NS	30.2 ± 2.5
CREAT (mg/dL)	1.4 ± 0.1	NS	1.39 ± 0.09
PAM (mm Hg)	46.2 ± 1.8	0.0001	32.2 ± 2.2
PCWP (mm Hg)	27.7 ± 1.8	0.002	18.9 ± 1.8
TPG (mm Hg)	18.2 ± 1.0	0.003	13.4 ± 1.0
CO (L/min)	3.6 ± 0.2	0.03	4.2 ± 0.15
PVR (WU)	5.1 ± 0.4	0.003	3.3 ± 0.3
MAP (mm Hg)	85.0 ± 3.6	0.03	75.7 ± 2.0
CVP (mm Hg)	11.7 ± 1.9	0.017	5.7 ± 1.3
SVR (WU)	21.2 ± 1.3	0.02	16.6 ± 1.1
Weight (kg)	84.6 ± 32.6	NS	78.9 ± 3.4

BUN = blood urea nitrogen; CO = cardiac output; CREAT = creatinine; CVP = central venous pressure; MAP = mean arterial pressure; NS = not significant; PAM = mean pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; TPG = transpulmonary gradient; WU = Woods units.

survival was also greater in group 1 (20 of 22; 91%) than in group 2 (10 of 16; 63%; $p < 0.05$). There were no perioperative deaths in patients who received transplants in either group.

Analysis of all data was performed using paired Student's *t* test and analysis of variance. There was no significant difference between the two groups in preoperative level of blood urea nitrogen or creatinine. There was no significant difference in any pretreatment variable between the two groups (Table 3). Table 4 compares hemodynamic variables for group 1 before and after therapy. There was a significant decrease in pulmonary arterial pressure, pulmonary capillary wedge pressure, and PVR, as well as a significant increase in CO. The preload (central venous pressure [CVP]) and afterload (systemic vascular resistance) were also reduced, but did not attain statistical significance. Table 5 compares pretreatment and posttreatment results for group 2 (conventional therapy). Pulmonary arterial pressure, pulmonary capillary wedge pressure, and PVR also decreased significantly, and there was a significant increase in CO. The preload (CVP) and afterload (systemic vascular resistance) were significantly reduced in this group. Comparison of the posttreatment variables between the two groups established that the PVR reduction in group 1 was significantly better than that of group 2. In no other measured variable was there a significant difference after therapy.

Table 4. Amrinone Therapy (n = 22)

Hemodynamic Variable	Pretherapy	*p* Value	Posttherapy
BUN (mg/dL)	28.8 ± 2.3	NS	30.2 ± 1.6
CREAT (mg/dL)	1.3 ± 0.06	NS	1.39 ± 0.07
PAM (mm Hg)	44.3 ± 1.6	0.0001	29.0 ± 1.5
PCWP (mm Hg)	25.2 ± 1.1	0.0001	18.5 ± 1.2
TPG (mm Hg)	19.0 ± 1.2	0.0001	10.9 ± 0.9
CO (L/min)	3.5 ± 0.3	0.015	4.56 ± 0.3
PVR (WU)	5.87 ± 0.4	0.0001	2.49 ± 0.2
MAP (mm Hg)	79.0 ± 3.4	NS	80.3 ± 3.0
CVP (mm Hg)	12.5 ± 1.9	NS	8.3 ± 1.8
SVR (WU)	20.8 ± 2.1	NS	16.4 ± 1.2

BUN = blood urea nitrogen; CO = cardiac output; CREAT = creatinine; CVP = central venous pressure; MAP = mean arterial pressure; NS = not significant; PAM = mean pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; TPG = transpulmonary gradient; WU = Woods units.

Analysis of the data from both groups was performed comparing those patients who responded to either therapy versus those who did not benefit from therapy (ie, patients who did not have a decrease in TPG, PVR, or PHT during treatment). Between the "responders" and "nonresponders," the only pretreatment variable of significance was CO. Mean CO in responders was 3.92 L/min, and in nonresponders, it was 3.07 L/min. The survival of all patients from both groups was compared. The only pretreatment variable with a significant difference was CVP. Mean CVP of survivors was 7.7 mm Hg versus 19.3 mm Hg in nonsurvivors.

Two of 22 patients (9%) in group 1 had a decrease in platelet count to less than 30,000/mL. Both patients had an initial count greater than 200,000/mL, but it decreased slowly between six and eight days and both patients required transfusion. The decrease was not transient; 1 patient received three separate platelet transfusions and the second patient was transfused at the time of transplantation. Both patients were weaned successfully off amrinone after transplantation, neither had serious postoperative bleeding requiring reoperation, and their platelet counts returned to more than 200,000/mL within ten days.

Comment

The presence of PHT and elevated PVR is a contraindication to orthotopic cardiac transplantation. Elevated PVR causes acute right heart failure in the transplanted heart secondary to an increased right ventricular afterload. Eichhorn and associates [4] showed the effects of phosphodiesterase inhibitors on right ventricular function using simultaneous radionuclide-hemodynamic studies. The phosphodiesterase inhibitor caused a 24% increase in cardiac index without a change in heart rate. The right ventricular preload was unchanged, and the ejection fraction was increased. With the increase in cardiac index and ejection fraction, the pulmonary arterial pressure decreased, indicating a substantial right ventricular afterload reduction. In a previous study, Bolling and associates [3] demonstrated a significant reduction in TPG, PVR, and pulmonary artery mean pressure using prolonged intravenous amrinone therapy in precardiac transplantation candidates. Cardiac output increased significantly, yet there was no significant decrease in systemic arterial pressure or vascular resistance. These data support the concept that amrinone has a direct effect in reducing right ventricular afterload.

Our data compare the hemodynamic changes between two groups of differently treated patients with end-stage congestive heart failure and PHT. Both therapies were effective in lowering the pulmonary hemodynamic values to a suitable range for transplantation. Conventional therapy (group 2) had significant effects on the systemic hemodynamics, causing both preload and afterload reduction on the heart. The subsequent pulmonary hemodynamic changes may be secondary to the systemic effects. Group 1 (amrinone) patients did not have significant changes in their systemic hemodynamics, yet manifested pulmonary hemodynamic changes suggesting primary action on the pulmonary vasculature.

Terris and colleagues [5] have shown that phosphodiesterase inhibitors have significant systemic unloading effects secondary to peripheral vasodilation. Furthermore, other investigators [6] have shown that the effects of phosphodiesterase inhibitors are more vasodilatory than inotropic, because left ventricular end-systolic pressure–diameter relation did not change when cardiac index improved and SVR decreased significantly. The discrepancy between our data concerning SVR and cardiac output and the data of others may reflect peripheral systemic hemodynamic compensation due to prolonged intravenous therapy. The initial systemic effects observed by Miller and co-workers [6] may not be consistent with prolonged therapy, yet favorable pulmonary hemodynamic changes may become evident over time. The pulmonary hemodynamic changes are not reflected by changes in total body water because body weight did not change significantly, although redistribution of body water from the lungs may have occurred. Amrinone-treated patients have a decrease in systemic vascular resistance and a subsequent rise in CO. Although this did not reach statistical significance, it may have contributed partially to the changes in pulmonary hemodynamics in the amrinone group. However, comparison of the systemic and pulmonary hemodynamic variables in both groups after therapy showed that the only variable with statistical significance was PVR. Even though both groups had significant reductions in the PVR, that of the amrinone group decreased more in comparison to that of the conventional group ($p < 0.05$), which supports a direct vasodilatory effect of amrinone on the pulmonary vasculature.

Analysis of the data of all patients in an effort to separate responders from nonresponders showed that pretreatment CO was the only significantly different variable (responders: CO = 3.91 L/min; nonresponders: CO = 3.09 L/min). Cardiac output, therefore, may have some predictive value in separating responders from nonresponders. Similarly, in assessing the differences between patients who survive until transplantation and patients who would not, the pretreatment CVP was significantly different (survivors: mean CVP = 8 mm Hg; nonsurvivors: mean CVP = 19 mm Hg) and may be of predictive value.

In conclusion, both conventional and amrinone therapy were effective in lowering pulmonary pressure and PVR to an acceptable range for orthotopic heart transplantation. Preoperative survival was significantly higher in the amrinone-treated group, possibly because these patients were in a hospital setting and were monitored closely. There was also a significant difference between the time interval of treatment and actual transplantation (group 1 = 27 days, group 2 = 8.5 months). Group 1 deaths were most often a result of progressive congestive failure, whereas in group 2 patients, sudden death, possibly due to volume contraction and hypokalemic-induced arrythmias, was more common. There were no perioperative deaths in the patients who received transplants in either group. Arminone achieved a more effective rate of response but required prolonged continual intravenous therapy. Pulmonary hypertension redeveloped in some patients once intravenous therapy was discontinued, and such patients therefore required continual in-hospital treatment.

Currently the average wait for a donor heart is 8 months, making continuous intravenous amrinone therapy impractical. Because conventional therapy poses a significantly greater risk of pretransplantation death, outpatient oral phosphodiesterase inhibitor therapy may be appropriate. A randomized prospective study comparing conventional therapy to outpatient oral phosphodiesterase therapy is indicated.

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BOOKS RECEIVED

The Works of William Harvey

Translated by Robert Willis, introduction by Arthur C. Guyton
Philadelphia, University of Pennsylvania Press, 1989
624 pp, not illustrated

This volume from the University of Pennsylvania Press includes the very famous *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus*. It also includes his book, *Anatomical Exercises on the Generation of Animals*, on which Harvey spent two thirds of his life. There is an excellent introduction to this book by Guyton, and the Robert Willis translations from the original Latin, which were made in 1847 and are considered the most accurate, have been used. This is a book that cardiothoracic surgeons will find interesting. The introduction by Dr Guyton is as lucid and informative writing as one finds in his books on physiology.

Interventional Cardiology

Edited by David R. Holmes, Jr, and Ronald E. Vlietstra
Philadelphia, F.A. Davis, 1988
400 pp, illustrated, $70.00

This is a monograph from the Department of Cardiology at the Mayo Clinic that describes their experience, and a short chapter by Dr Hartzell Schaff on the role of the surgeon in interventional cardiology. This clearly gives the viewpoint of the Mayo Clinic.

De Vega's Annuloplasty for Acquired Tricuspid Disease: Early and Late Results in 110 Patients

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From January 1978 through February 1989, 110 tricuspid annuloplasties (De Vega's procedure) were performed in association with mitral and combined mitral and aortic valve disease. Preoperatively, 106 (96%) of 110 patients were in New York Heart Association functional class III or IV. There were seven early deaths (6.3%), and 3 patients, 2 with mitral lesions and 1 with a combined lesion, died during a follow-up period of 3 to 52 months (mean follow-up, 22 months). Four patients (3.6%) required reoperation because of biological mitral valve failure at 5 to 8 years after tricuspid annuloplasty (mean period, 6.6 years). Twenty-three (62%) of 37 randomly selected patients evaluated by echocardiography and 14 (70%) of 20 patients evaluated by right ventriculography showed complete disappearance of tricuspid regurgitation after tricuspid annuloplasty in 1 to 18 months (mean period, 3.3 months). Seventy-seven (96%) of the survivors were in functional class I or II after tricuspid annuloplasty. The actuarial survival rate for the TAP series including early deaths was 85.8% ± 7.4% at 10 years and the actuarial rate of freedom from reoperation on the tricuspid valve was 96.7% ± 1.4%. Our surgical experience indicates that the De Vega's annuloplasty, as the method of first choice, is a simple, reliable procedure and resulted in improvement in 90% of patients with moderate to severe functional tricuspid regurgitation.

(Ann Thorac Surg 1989;48:670–6)

Surgical correction of acquired tricuspid regurgitation (TR) with long-standing chronic mitral or aortic valve disease, or both, is still controversial. In most instances this TR is functional secondary to pulmonary hypertension and right ventricular dilatation. Therefore, according to some authors [1], TR should regress spontaneously after repair of the mitral lesion. However, the application of pulsed Doppler echocardiography provides us an accurate diagnosis of the TR before and after operation, and suggests the necessity for repair of residual TR, which otherwise would worsen the postoperative course even after the mitral repair [2, 3].

In this study, we evaluated the efficacy of tricuspid annuloplasty (TAP) (De Vega's procedure [4]) in 110 patients with tricuspid valve disease by echocardiography, postoperative hemodynamics, clinical status, and long-term results.

Material and Methods

Between January 1978 and February 1989, 685 patients underwent a mitral or combined mitral and aortic valve operation at Sapporo Medical College Hospital. Of these patients, 128 (19%) required surgical treatment of moderate to severe tricuspid valve disease. The TAP of De Vega was performed in 110 patients and tricuspid valve replacement (TVR) in 18 patients, 4 of whom had had previous TAP and mitral valve replacement (MVR).

Patient Population

There were 42 male and 68 female patients among the 110 patients who had TAP. They ranged in age from 13 to 69 years with a mean age of 57 ± 7 years. Preoperatively, 4 patients (4%) were in New York Heart Association functional class II, 61 (55%) were in class III, and 45 (41%) were in class IV. Eighty-four (76%) of the 110 patients had mitral stenosis, and 26 (24%) had mitral regurgitation.

Forty-two (38%) of the 110 patients had undergone previous cardiac procedures: MVR, 23; open mitral commissurotomy 17; aortic valve replacement (AVR) + MVR, 2; and AVR + open mitral commissurotomy, 2.

All of the patients underwent preoperative cardiac catheterization and angiography. The severity of TR before and after operation was estimated by right ventriculography, pulsed Doppler echocardiography, and two-dimensional echocardiography.

The indication for TAP or TVR was determined by the following criteria: (1) mean right atrial pressure of 10 mm Hg or more at rest; (2) TR of grade 3/4 or more and end-systolic tricuspid annulus diameter of 35 mm or more by right ventriculography in the 30-degree right anterior oblique projection; (3) TR distance from the tricuspid annulus of 30 mm or more as estimated by pulsed Doppler echocardiography; (4) TR area of 400 mm^2 or more as calculated by pulsed Doppler echocardiography; (5) moderate to severe TR by digital evaluation before the institution of cardiopulmonary bypass; and (6) extent of valve competence as determined by the injection of cold saline solution through a pigtail catheter from the main pulmonary artery into the right ventricle. If one or more of

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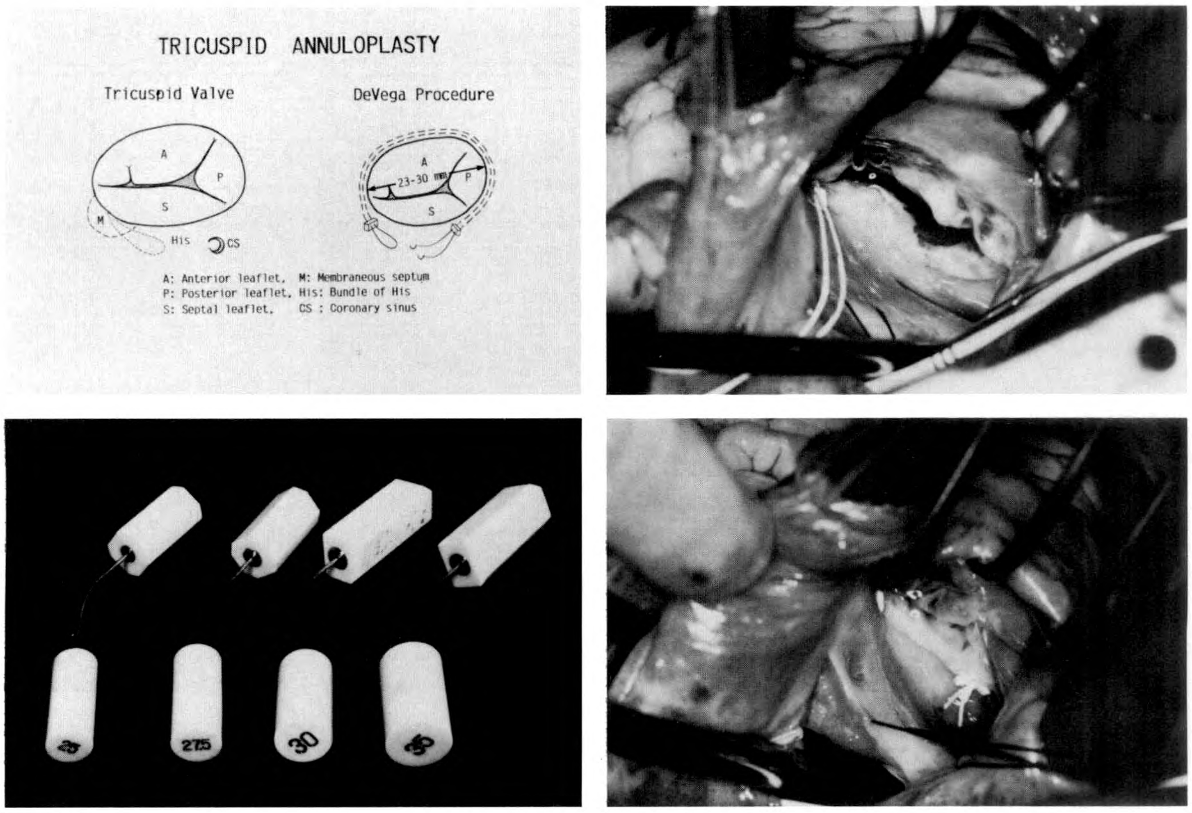

TRICUSPID ANNULOPLASTY
Tricuspid Valve
DeVega Procedure
A
P
S
M
His
CS
23-30 mm
A: Anterior leaflet, M: Membraneous septum
P: Posterior leaflet, His: Bundle of His
S: Septal leaflet, CS : Coronary sinus
25
275
30

Fig 1. A Gore-Tex suture with Teflon felt was started at the commissure between the septal leaflet and the anterior leaflet and ended at the area between the posterior leaflet and the septal leaflet. The annulus diameter of the tricuspid valve was reduced to normal size (mean diameter, 26.4 ± 2.7 mm; range, 23 to 30 mm) by use of sizers (bottom left) developed by us. Before (top right) and after (bottom right) the De Vega annuloplasty, the saline solution injection test using a pigtail catheter from the main pulmonary artery into the right ventricle was done to confirm valve competence.

these criteria were present, TAP was indicated; if there was marked TR after TAP, then TVR was performed.

Operative Procedure

All patients had a median sternotomy, and the heart was arrested with cold crystalloid or blood cardioplegia. After the mitral or the combined mitral and aortic valve repairs were performed, the right atrium was opened and the tricuspid valve was examined. In patients with moderate to severe TR, valve competence was determined by the saline solution injection method, and if there was marked regurgitation from the leaflets and the dilated annulus was more than 35 mm in size, annuloplasty was indicated.

The annuloplasty was done as follows (Fig 1): One 2-0 polypropylene or Gore-Tex suture with a Teflon felt pledget was started at the commissure between the septal and anterior leaflets and ended at the commissure between the posterior and septal leaflets. The diameter of the annulus was reduced to 23 to 30 mm (mean size, 26.4 ± 2.7 mm) with a sizer we developed.

After completion of the annuloplasty, the saline solution injection test was done again to confirm valve competence. If there was still marked regurgitation from the center of the leaflets, TVR was indicated. Eighteen of the 128 patients with tricuspid valve disease underwent TVR, and 4 of them were among the 110 who had had a De Vega procedure. In these 4, TVR was performed 5 to 8 years (mean period, 6.5 years) after TAP.

The cumulative follow-up for patient's having a De Vega annuloplasty was 487.8 patient-years with a range of 2 months to 11.2 years (mean follow-up, 4.2 years).

Statistical Analysis

All data are expressed as the mean ± the standard deviation. Actuarial survival analysis was calculated by the method of Kaplan and Meier.

Results

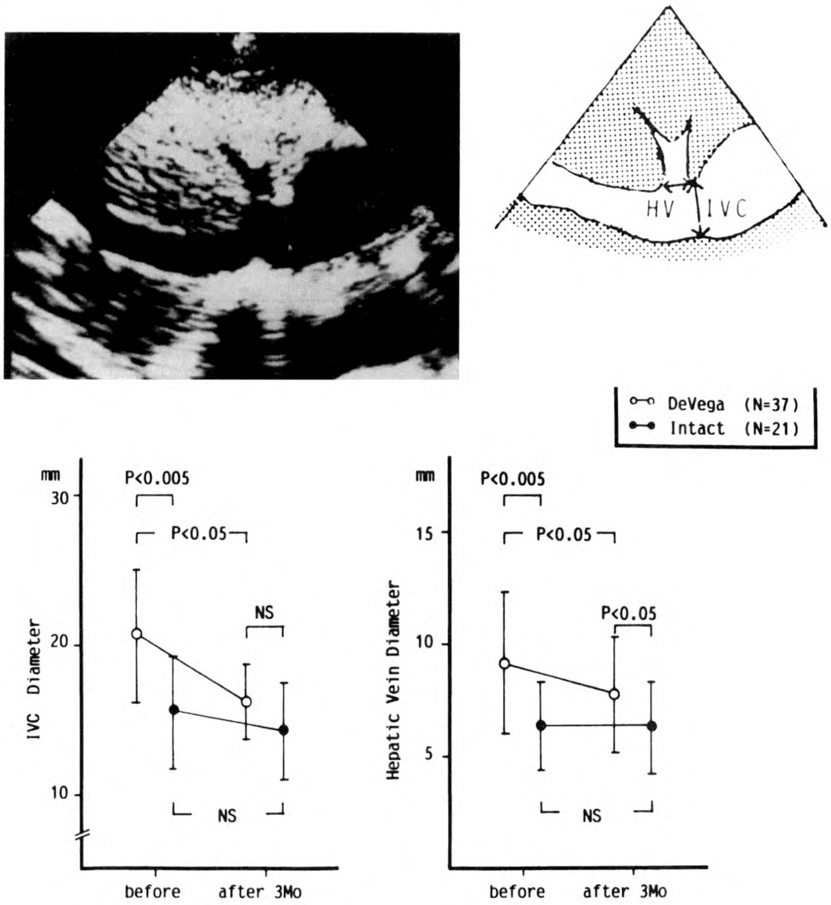
Early Deaths

There were seven early deaths (6.3%) among the 110 patients undergoing TAP. There was no significant difference in early mortality between the mitral and combined mitral and aortic groups. The causes of early death were related to preoperative myocardial failure in 4 patients, hemorrhage due to left ventricular rupture in 1 patient, arrhythmia in 1, and respiratory failure in 1.

Late Deaths

Three patients (2.7%), 2 with mitral lesions and 1 with a combined lesion, died during a follow-up of 3 to 52

Fig 2. Changes in diameter of inferior vena cava (IVC) and hepatic vein (HV) before and after operation with and without a De Vega annuloplasty. (NS = not significant.)



months (mean period, 22 months). The causes of death were myocardial failure, cerebral embolism, and sudden death.

Reoperation

Four patients required reoperation after the De Vega operation. These patients had severe TR due to failure of the biological mitral valve (two Hancock and two Liotta biological valves). At operation, the tricuspid annulus was not enlarged, but the leaflets were degenerated and the chordae were elongated with marked regurgitation from the center of the leaflets. These valves were replaced with mechanical valves (St. Jude Medical).

Echocardiographic Evaluation

The efficacy of the De Vega procedure was evaluated by pulsed Doppler echocardiography in 37 randomly selected patients and 21 patients who had not undergone TAP for TR because of mild to moderate TR before mitral repair. In 23 (62%) of the 37 patients, the TR had completely disappeared; 11 patients (30%) had mild TR (grade 1–2/4), and 3 patients (8%) still had moderate TR (grade 3/4) postoperatively. Each measurement was done using two-dimensional and pulsed Doppler echocardiography.

The tricuspid annulus was significantly reduced from 45.8 ± 7.8 mm to 28.4 ± 1.1 mm after the De Vega procedure, but when this procedure was not done, there was no significant change in diameter (preoperative valve, 36.7 ± 7.5 mm, and postoperative valve 34.7 ± 6.4 mm). The changes in diameter of the inferior vena cava and hepatic vein are shown in Figure 2. The preoperative mean diameter of the inferior vena cava was 20.4 ± 4.5 mm; after TAP, this had decreased significantly to a mean of 16.7 ± 2.7 mm ($p < 0.01$). However, without TAP, the mean value did not decrease even after repair of the mitral valve. Similar observations were made for the diameter of the hepatic vein.

The changes in TR distance and TR area of systolic turbulence from the tricuspid valve by pulsed Doppler echocardiography are shown in Figure 3. The mean preoperative TR distance of 42.6 ± 12.2 mm in patients having TAP was significantly longer than the mean value of 22.9 ± 7.6 mm in patients with intact TR ($p < 0.001$). The TR distance was significantly decreased with TAP (8.5 ± 10.2 mm) ($p < 0.01$), but in patients who did not have TAP, the mean TR distance was not improved (mean value, 22.4 ± 13.2 mm). The mean TR area (554 ± 260 mm^2) before operation in patients who had TAP was significantly larger than the mean value in those with intact TR (152 ± 9.5 mm^2) ($p < 0.01$). With TAP, the TR area was significantly decreased to 58 ± 70 mm^2 ($p < 0.005$); but without TAP, this value was unchanged (130 ± 13 mm^2).

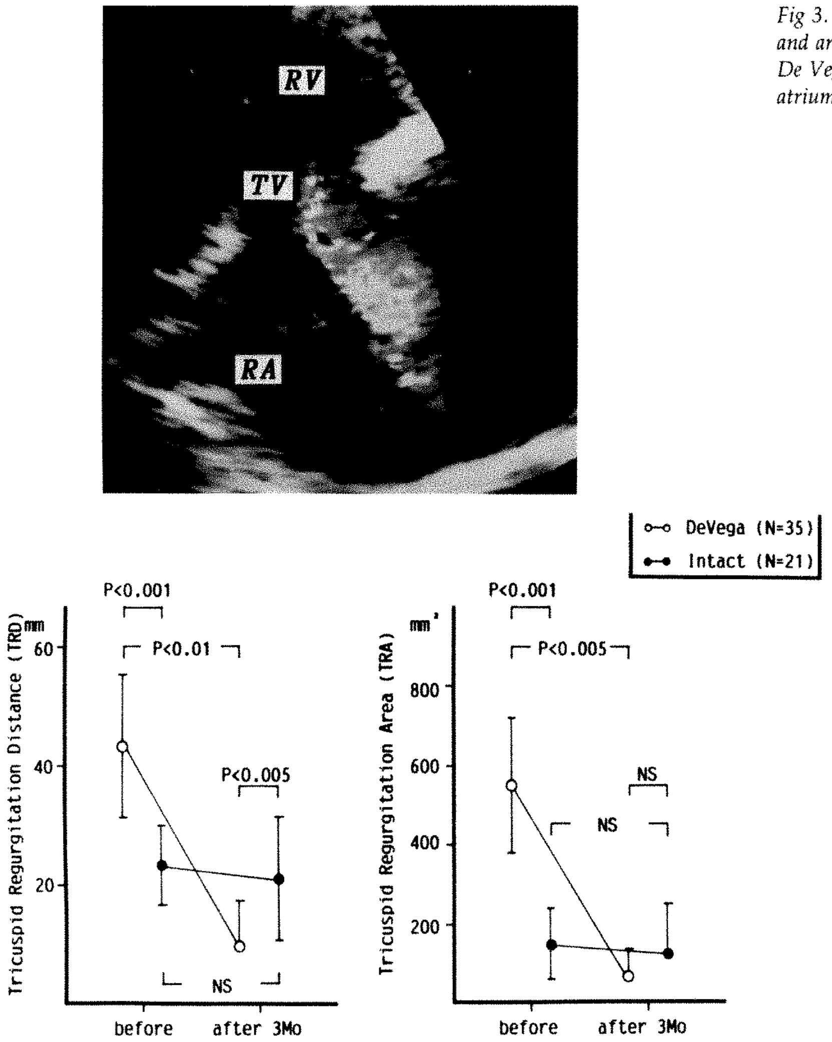


Fig 3. Changes in tricuspid regurgitation (TR) *distance and area before and after operation with and without a De Vega procedure.* (NS = *not significant;* RA = *right atrium;* RV = *right ventricle;* TV = *tricuspid valve.)*

Hemodynamic Evaluations

The mean preoperative right atrial V wave in patients having TAP (9.7 ± 4.1 mm Hg) was higher than in patients not having TAP (4.3 ± 1.8 mm Hg). The mean value decreased significantly with TAP (5.6 ± 1.8 mm Hg) ($p < 0.01$), but was unchanged without TAP (4.2 ± 1.6 mm Hg).

Right ventriculograms in the 30-degree right anterior oblique projection were obtained for comparison with the findings on pulsed Doppler echocardiography before and after operation in 32 patients with and without a De Vega annuloplasty. The TR in these patients was graded as follows: mild (1–2/4) (small jet or mild TR) in 12 patients (37.5%); moderate (3/4) (contrast medium in the right atrium) in 16 (50%); and severe (4/4) (contrast medium in both the right atrium and the inferior vena cava) in 4 (12.5%). The 20 patients with moderate to severe TR had a De Vega operation after mitral and combined mitral and aortic operations. The TR disappeared completely in 14 patients (70%), remained mild in 4 (20%), and remained moderate in 2 (10%). These results correlated quite well with the findings on pulsed Doppler echocardiography after TAP.

Twenty-eight patients (13, TAP + MVR; 8, TAP + AVR + MVR; and 7, intact TR + MVR) underwent cardiac catheterization and cardiac function studies before and after operation (range, 1 month to 18 months; mean, 3.3 months) (Fig 4). The preoperative mean right atrial pressure in patients having TAP + MVR and TAP + AVR + MVR was significantly higher than in patients with intact TR + MVR. In the TAP groups, these values were significantly decreased postoperatively. Mean pulmonary artery pressure and mean pulmonary capillary wedge pressure in the TAP + AVR + MVR and TAP + MVR groups were significantly decreased after operation. There was no significant difference in mean cardiac index before operation in the three groups. After operation, this index increased significantly in the TAP groups, but not in the patients who did not undergo TAP.

Clinical Status

The cardiothoracic ratio decreased significantly from preoperative mean values of 62.7% ± 9.4% in the group with intact TR + MVR, 72.4% ± 6.3% in the group having TAP + MVR, and 73.4% ± 8.4% in the group having TAP + AVR + MVR to postoperative mean values of 58.4% ±

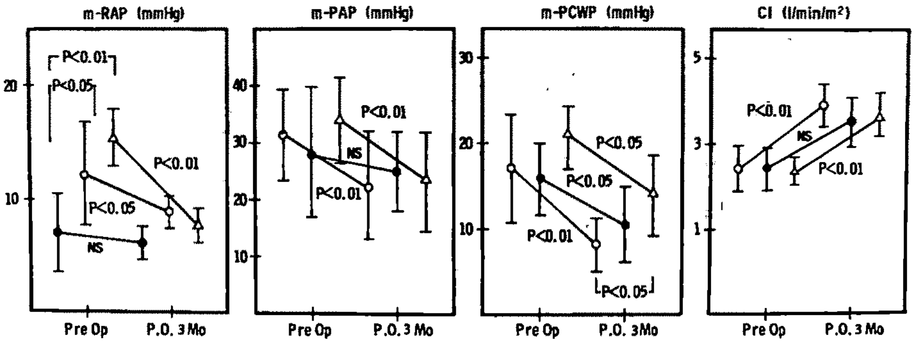


Fig 4. Hemodynamics in 13 patients who underwent mitral valve replacement (MVR) *and a De Vega annuloplasty* (TAP) (O-O), *in 8 patients who underwent aortic valve replacement* (AVR), *and TAP* (Δ-Δ), *and in 7 patients who had MVR without TAP* (●-●) *before and 3 months after operation.* (CI = *cardiac index;* m-PAP = *mean pulmonary artery pressure;* m-PCWP = *mean pulmonary capillary wedge pressure;* m-RAP = *mean right atrial pressure;* NS = *not significant.)*

8.7%, 63.1% ± 2.9%, and 64.1% ± 3.7%, respectively ($p < 0.05$, $p < 0.01$, and $p < 0.001$), respectively).

New York Heart Association functional class after TAP for 80 survivors followed long term was as follows: 44 (55%) in class I, 33 (41%) in class II, and 3 (4%) in class III. The severity of the preoperative functional class in the TAP + MVR + AVR group was significantly greater than in the group with intact TR + MVR. The mean values for each group were significantly improved after operation. However, after TVR, the 16 survivors (89%) were classified as follows after the same follow-up: 10 patients (62.5%) in class I; 4 (25%) in class II; and 2 (12.5%) in class III.

The actuarial survival rate including early deaths after a De Vega procedure was 85.8% ± 7.4% at 10 years; it was 87.5% ± 4.8% for the TAP + MVR group and 86.7% ± 8.7% for the TAP + AVR + MVR group (Fig 5). However, after TVR, this rate was 83.3% ± 11.4% at 4.4 years. There were no significant differences between the three groups.

Comment

Patients with long-term chronic mitral and combined mitral and aortic disease can have TR, and in such patients, severe TR sometimes worsens the surgical results after the mitral operation and the prognosis [2, 3]. The incidence of acquired TR among patients with mitral disease ranges from 22% to 30% [2, 3, 5, 6]. In most instances, TR was thought to have occurred in patients with pulmonary hypertension caused by mitral valve lesions, and therefore right ventricular enlargement might be involved in the pathogenesis of functional TR [5]. According to Starr [7], right ventricular–tricuspid valvular disproportion with compromised function of the tricuspid valve apparatus, rather than dilatation of the annulus of the tricuspid valve, is an important component in functional TR. Simon and colleagues [3] reported that a depressed extent of tricuspid annulus shortening in systole seems to be important in the pathogenesis of TR. They also thought that besides hemodynamic factors secondary to mitral disease, a myocardial factor may be involved in the pathogenesis of persistent TR.

In the present study, 110 (86%) of 128 patients had a De Vega annuloplasty and the remaining 18 patients (14%), TVR. There was no significant difference in early mortality (TAP, 7/110 or 6.3%, and TVR, 1/18 or 5.5%) or late mortality (TAP, 3/110 or 2.7%, and TVR, 1/18 or 5.5%).

Clinically, evident functional TR disappears or masks itself after intensive medical treatment, and it is sometimes difficult to accurately assess the severity of the lesion and to decide on the surgical repair. However, the newer techniques of two-dimensional and pulsed Doppler echocardiographic measurements of the TR fraction have allowed objective, reproducible, noninvasive diagnosis of tricuspid valve motion, regurgitation, and venous dilatation [8]. These techniques can also demonstrate the necessity for TR repair after the mitral or aortic and mitral operation. Surgical repair of moderate to severe TR is

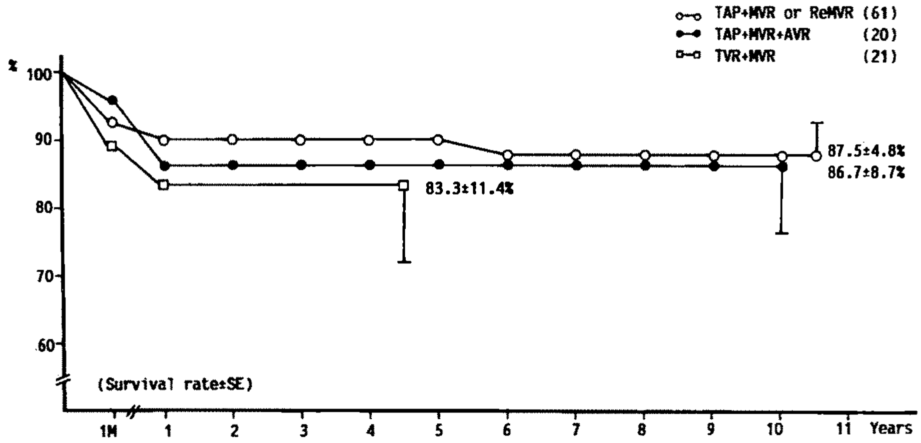


Fig 5. Actuarial survival rate after a De Vega annuloplasty (TAP) + *mitral valve replacement* (MVR) *or repeat MVR, after TAP + MVR + aortic valve replacement* (AVR), *and after tricuspid valve replacement* (TVR) + *MVR.* (SE = *standard error.)*

necessary in a number of patients to obtain a better prognosis. Also, recent improvements in precise preoperative diagnosis and techniques of myocardial protection have brought about better surgical procedures.

We decided the De Vega procedure was indicated for those patients with one or more of our criteria before operation, and we also decided on TVR for patients with severe organic changes in the tricuspid leaflets and dilatation of the annulus after TAP.

At operation, the decision of whether to repair or replace is crucial. Tricuspid annuloplasty as described by De Vega [4] is a simple and short procedure, and it does not disturb the pathway of the His bundle or the valve architecture. An early mortality rate of 4% to 31% has been reported [3, 4], but recent myocardial protection techniques have reduced the number of early deaths.

In our TAP series, there were 7 early deaths (6.3%), and most were due to preoperative myocardial failure resulting from severe mitral lesions or primary tissue failure of biological mitral valves. There were no deaths related to the annuloplasty itself. The ideal annuloplasty is easy to perform, and the technique should be reproducible, preserve normal valve function, lead to predictable results, and have a long-lasting effect.

Three annuloplasty repairs [4, 9, 10] have been applied for functional TR. Bicuspidalization annuloplasty, described by Kay [9], Boyd [2], Nakano [11], and their co-workers, is a simple technique that converts the tricuspid valve into a bicuspid valve by exclusion of the posterior leaflet, and has a gratifying late result [11]. The Carpentier annuloplasty is performed to shorten the annulus with either rigid or flexible, circular or semicircular prosthetic rings [10]. Grondin and associates [12] reported that the De Vega annuloplasty and the Carpentier annuloplasty were superior to the bicuspidalization procedure in hemodynamic studies 10 and 11 months after operation. Peterffy and colleagues [13], however, reported that using the Kay annuloplasty and the De Vega annuloplasty, they obtained similar late results in about 70% of their patients after each type of repair.

In the present study, the efficacy of the De Vega annuloplasty was evaluated a relatively short period after operation. In 23 (62%) of 37 randomly selected patients evaluated by echocardiography, TR had disappeared. Eleven patients (30%) still had mild TR, and 3 patients (8%) still had moderate TR postoperatively. Similar results were obtained by right ventriculography in 20 patients with moderate to severe TR; 18 (90%) showed improvement after a De Vega annuloplasty.

Overrepair of TR with a De Vega procedure can lead to stenosis and underrepair, to residual regurgitation. These disadvantages may explain the postoperative hemodynamic results reported by Chidambaram [14], Simon [3], and their coauthors. In our surgical management, annuloplasty was performed to reduce the tricuspid orifice with a sizer, as illustrated in Fig 1, to a normal tricuspid orifice of 23 to 30 mm (mean size, 26.4 ± 2.7 mm) in diameter. Like Rowlatt and associates [15], we found no evidence of stenosis at postoperative hemodynamic and echocardiographic evaluations.

Tricuspid annuloplasty did not cause any deaths or morbidity in our patients. The causes were the severity and the duration of the mitral valve disease, and myocardial failure. Reoperation in 4 patients after annuloplasty was necessitated by failure of the biological mitral valve. The actuarial survival rate for the 110 patients after annuloplasty was 85.8% at 10 years, and the actuarial rate of freedom from reoperation on the tricuspid valve was 96.7% in our series.

Tricuspid valve replacement has been favored over annuloplasty by some surgeons [16, 17] who have reported that valve replacement is superior to annuloplasty because of the unsatisfactory results with annuloplasty. Thorburn and associates [18], however, reported a high incidence (20%) of thrombosed valves in 71 patients and a higher early mortality with TVR than TAP. Kratz and co-workers [19] reported that the actuarial survival rate at 8 years after TVR was 41% versus 62% after TAP.

In our series, 18 (14%) of 128 patients had TVR, including 4 at 5 to 8 years (mean period, 6.6 years) after TAP. All 18 of these patients except 1 had previously had open mitral commissurotomy or MVR. In our series, there were no significant differences in early and late mortality between TVR (1/18 or 5.5% early and 1/18 or 5.5% late) and TAP (7/110 or 6.3% early and 3/110 or 2.7% late). The advantages of TVR are that complete repair of the tricuspid valve in patients with severe congestive heart failure is performed and that early results are predictable; therefore, the valve does not become incompetent secondary to mitral valve failure.

In the present study, the actuarial survival rate after TVR in 18 patients was 83.3% at 4.4 years and the actuarial rate of freedom from reoperation was 100% at 4.4 years. Fourteen (78%) of the 18 patients were in functional class I or II postoperatively. However, although there were good hemodynamic findings and a good clinical status after TVR, this procedure has its own inherent morbidity from valve thrombosis [18], primary tissue failure of biological valves, and anticoagulant-related hemorrhage with mechanical valves.

In conclusion, our early and long-term results at 10 years in 110 patients who underwent a De Vega annuloplasty were good with low early and late mortality and morbidity. Our surgical experience suggests that the De Vega annuloplasty, as the method of first choice, resulted in improvement in 90% of patients with moderate to severe functional TR, and that TVR can also be safely applied to repair severe organic disease of the tricuspid valve.

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Replacement of the Esophagus by a Segment of Colon Provided With an Antireflux Valve

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A method has been developed to prevent reflux of gastric contents into an interposed segment of colon substituting for the esophagus. The operative procedure includes construction of an intussusception valve at the cologastric junction. The method was used in 5 patients, who have been followed clinically for 17 to 30 months. Clinical observations including endoscopy as well as radiography and dynamic scintigraphy indicate that the addition of an antireflux valve at the distal end of the interposed segment of colon prevents gastrocolic reflux without jeopardizing the emptying of the colon transplant.

(Ann Thorac Surg 1989;48:677–82)

The risk of reflux is an ever-present cause of concern when esophageal replacement is done by any method [1]. To date, no surgical method has been described that effectively prevents regurgitation of gastric contents to an interposed segment of colon. Although late complications are rare in conventional colon interposition, acid or biliary reflux to the transposed colon can cause ulcerations or stricture formations, especially at the anastomoses [2–5]. In this report, we describe a technique for prevention of gastrocolic reflux and present our clinical experience.

Material and Methods

Patient Population

Since September 1986, 5 patients have undergone operation by this method. The average age of the patients was 35 years (range, 17 to 64 years).

Four patients had a benign esophageal lesion and 1 patient, a tracheal rhabdomyosarcoma with involvement of the upper esophagus, pharynx, and larynx. One patient had undergone a Heller myotomy, an antireflux procedure, and a longitudinal plication of a gross megaesophagus, and then required many balloon dilations with only temporary relief of symptoms. The indication for operation in another patient, who previously had undergone colon replacement of the esophagus because of a benign stenosis, was serious reflux complications with recurrent severe bleeding and nocturnal aspirations. He had been operated on twice because of hiatal hernia and once because of an iatrogenic perforation with esophagorraphy. He had redundant interposed colon, the distal part of which was dissected free at reoperation and used for the construction of an intussusception valve.

Operative Technique

Resection of the esophagus and replacement with a segment of colon were performed through a separate upper abdominal midline incision and a right thoracotomy. One patient had a pharyngocolostomy through an additional cervical incision, and the others had a proximal intratho-

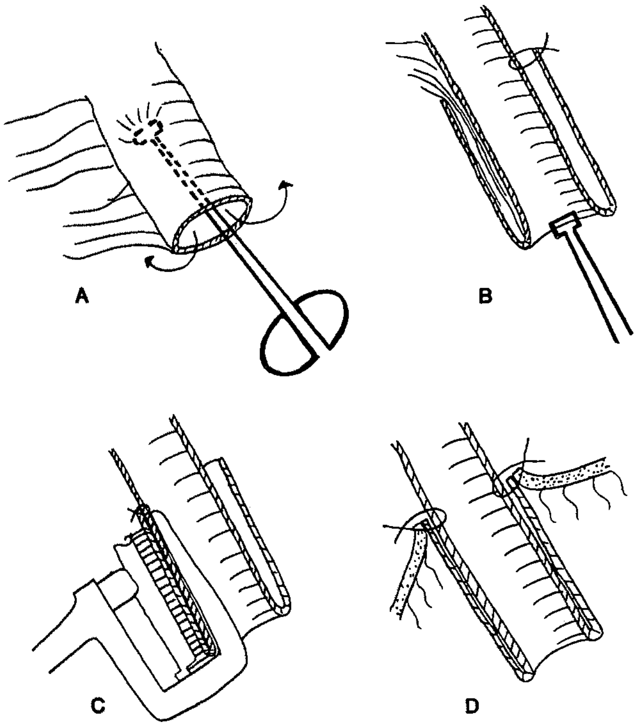

Fig 1. Construction of antireflux valve at cologastric junction. (A) By grasping the bowel wall through the open lumen with a Babcock's forceps, the terminal segment of the colon is turned inside out. (B) The position of the valve is secured by suturing the two leaves of the base with interrupted 4-0 Dexon sutures. (C) The intussuscepted position is maintained by application of two to three rows of staples longitudinally. (D) The valve is brought into the lumen of the stomach and anastomosed end-to-end to the cut edge of the cardia with one-layer suture technique.

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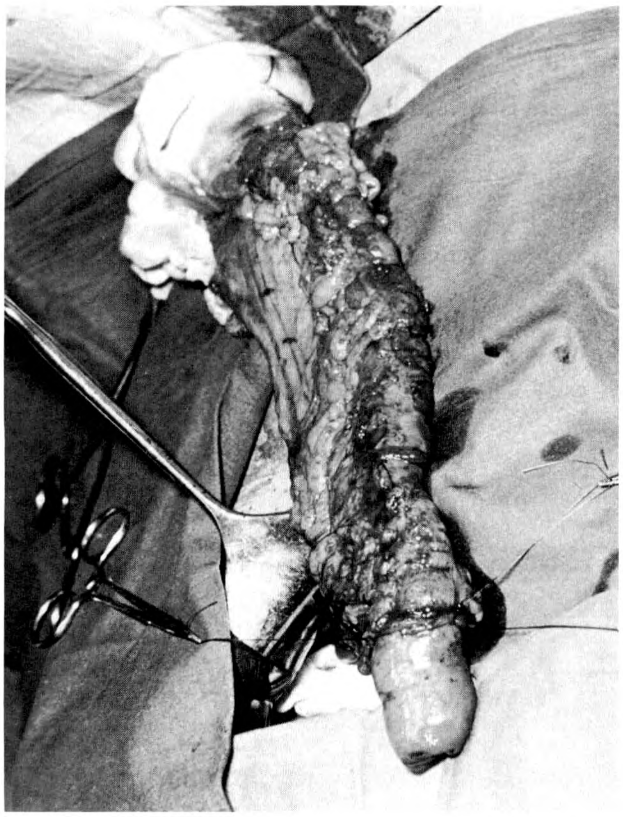

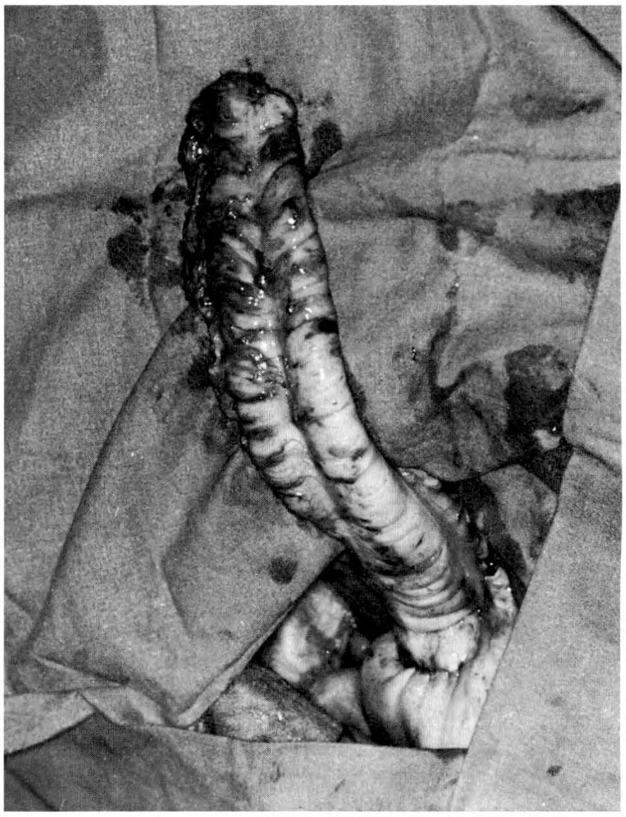

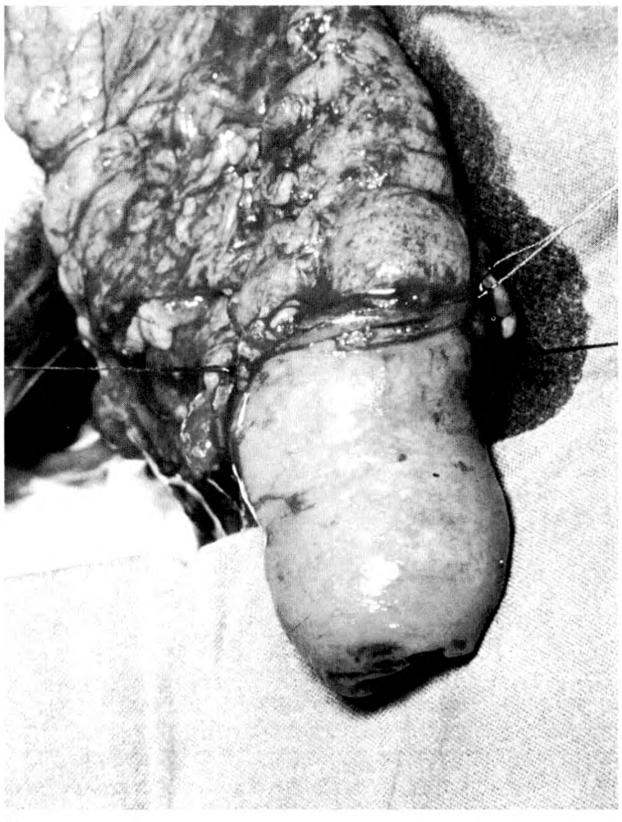

Fig 2. (A) A segment of transverse and descending colon has been prepared. An intussusception valve has been constructed at the distal end. (B) A closer view of the valve. (C) The end of the graft with the valve has been placed into the stomach, and the cologastric anastomosis has been completed.

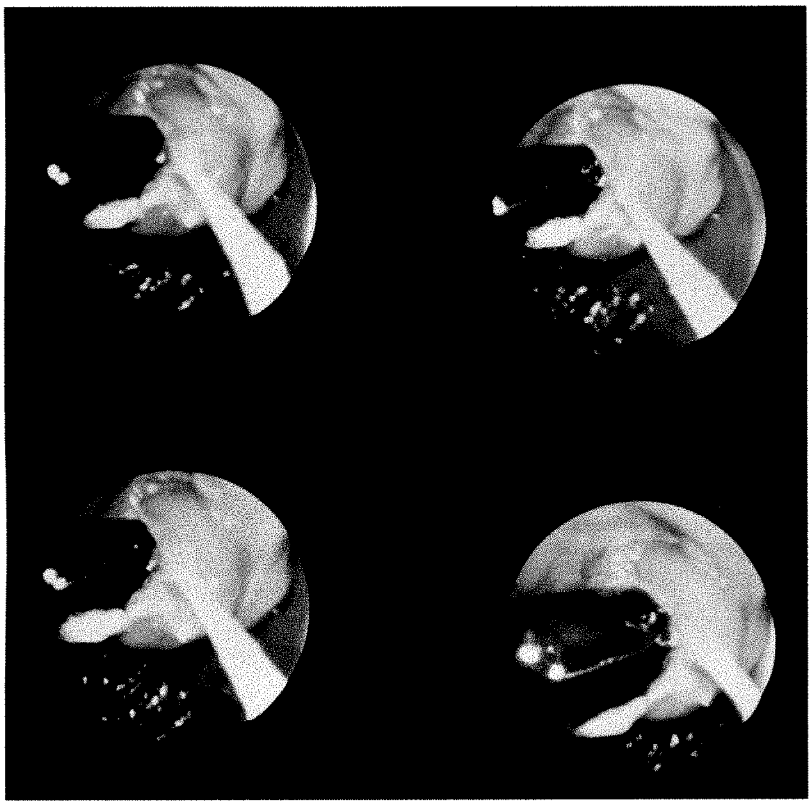

Fig 3. Endoscopic views of the valve as seen from the gastric cavity. The esophagoscope passed easily through the transposed colon and the valve into the stomach. The whitish band is mucus.

racic anastomosis. In 1 patient, who previously had undergone a conventional colon replacement of the esophagus, the antireflux procedure was carried out through an abdominal incision alone at a second operation.

A segment of the transverse and left colon supplied by the ascending branch of the inferior mesenteric artery and corresponding veins was isolated and placed in the isoperistaltic position in the bed of the resected esophagus. A retrogastric graft pedicle was employed. One-layer suture technique was used for the proximal end-to-end anastomosis to the esophagus or the pharynx.

Vagotomy, pyloromyotomy, and feeding jejunostomy were performed. One patient also had a gastrostomy.

The technique used for construction of the antireflux valve is demonstrated in Figures 1 and 2. The bowel wall was grasped through the open lumen with a Babcock's forceps and the terminal section of the interposed segment of colon was turned inside out. The length of the valve was 5 cm at least. The valve was secured by suturing the two leaves at the base with interrupted 4-0 Dexon. The intussusception was also stabilized by the application of three rows of staples longitudinally with an autosuture instrument. One row was placed at the antimesenteric border, and the other rows were between the mesenteric and antimesenteric borders on each side of the valve. Resection of the esophagus was carried out just distal to the esophagogastric junction. The valve was then brought into the lumen of the stomach and anastomosed end-to-end to the cut edge just below the cardia with interrupted one-layer suture technique. Stitches through all layers and Lembert sutures were placed alternately. Great care was taken not to compromise the vascular supply of the valve.

Results

The early postoperative course was completely uneventful in 3 patients. One week after operation, 1 patient had severe pneumonia and was maintained on a respirator for ten days. He now is well and has gained 8 kg. One patient returned to the hospital 1 month after operation with signs of mechanical ileus. The obstruction was caused by adhesions; adhesiotomy was successful.

One patient died 2 months postoperatively of widespread metastatic disease, although free from cancer locally in the neck. The other 4 patients have been followed carefully for 17 to 30 months. The clinical results have been evaluated, and the interposed segment of colon and the valve have been studied by endoscopy, radiography, and scintigraphy at regular intervals.

The clinical result was evaluated according to the criteria of Hanna and associates [6]. The result was excellent in 2 patients and good in the other 2, both of whom occasionally experience mild dumping syndrome. They have a normal ability to eat and have had no signs of reflux or problems with belching or foul breath. All 4 patients have expressed satisfaction with the procedure.

Endoscopic examination showed normal findings in the interposed segment of colon. No retention of food or regurgitation of gastric contents was observed. The valve was closed, but an instrument could easily pass through it. Endoscopic views of the valve as seen from the gastric cavity are shown in Figure 3.

A barium meal examination in 1 patient with a valve is shown in Figure 4. During fluoroscopy, the interposed segment of colon showed slow wall motions, and the contrast medium passed through the valve in portions.

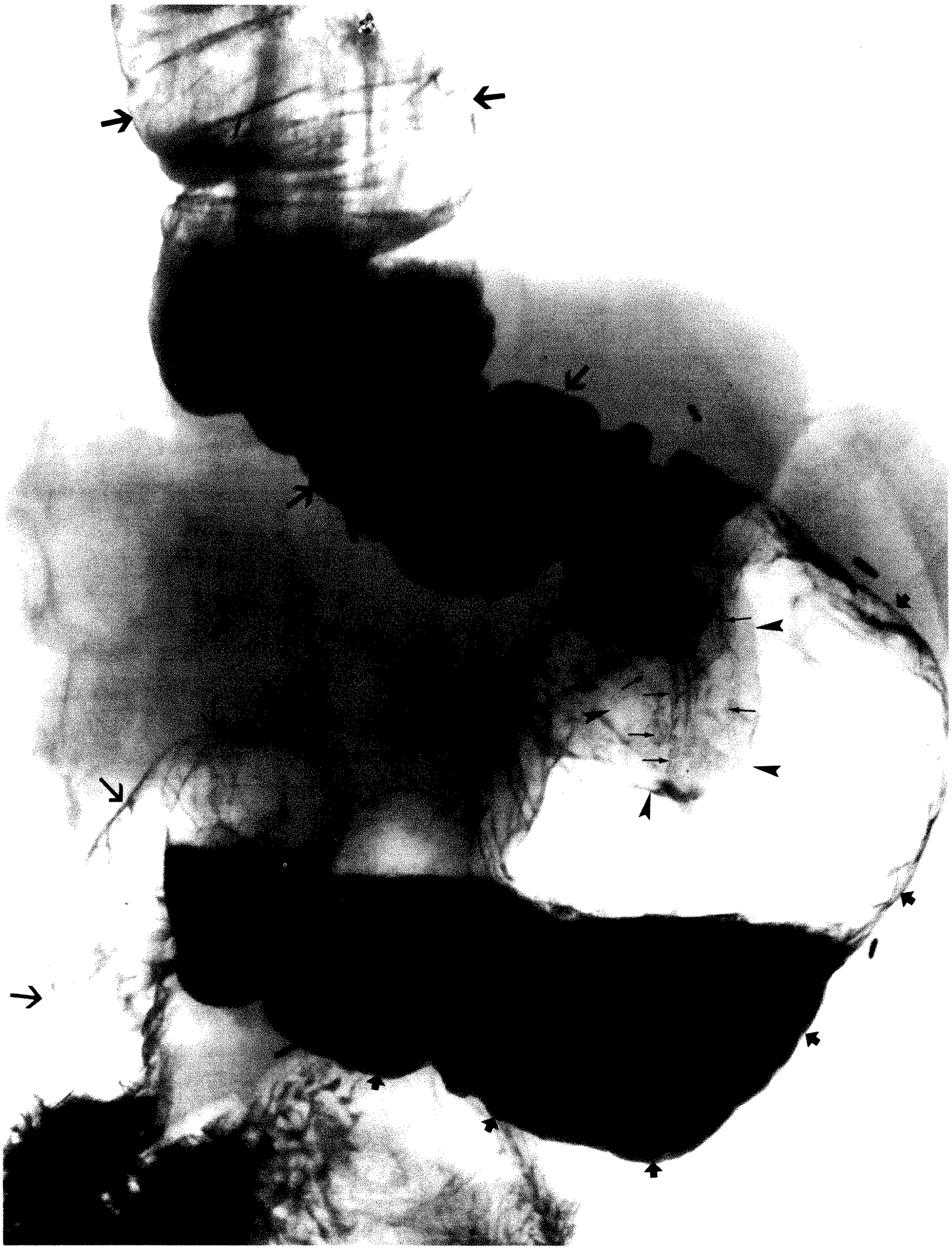

Fig 4. Interposed segment of colon (➔) with an antireflux valve (►) protruding into the stomach. Stabilizing staples (→), greater curvature of the stomach (➧), and duodenum (➔) are shown.

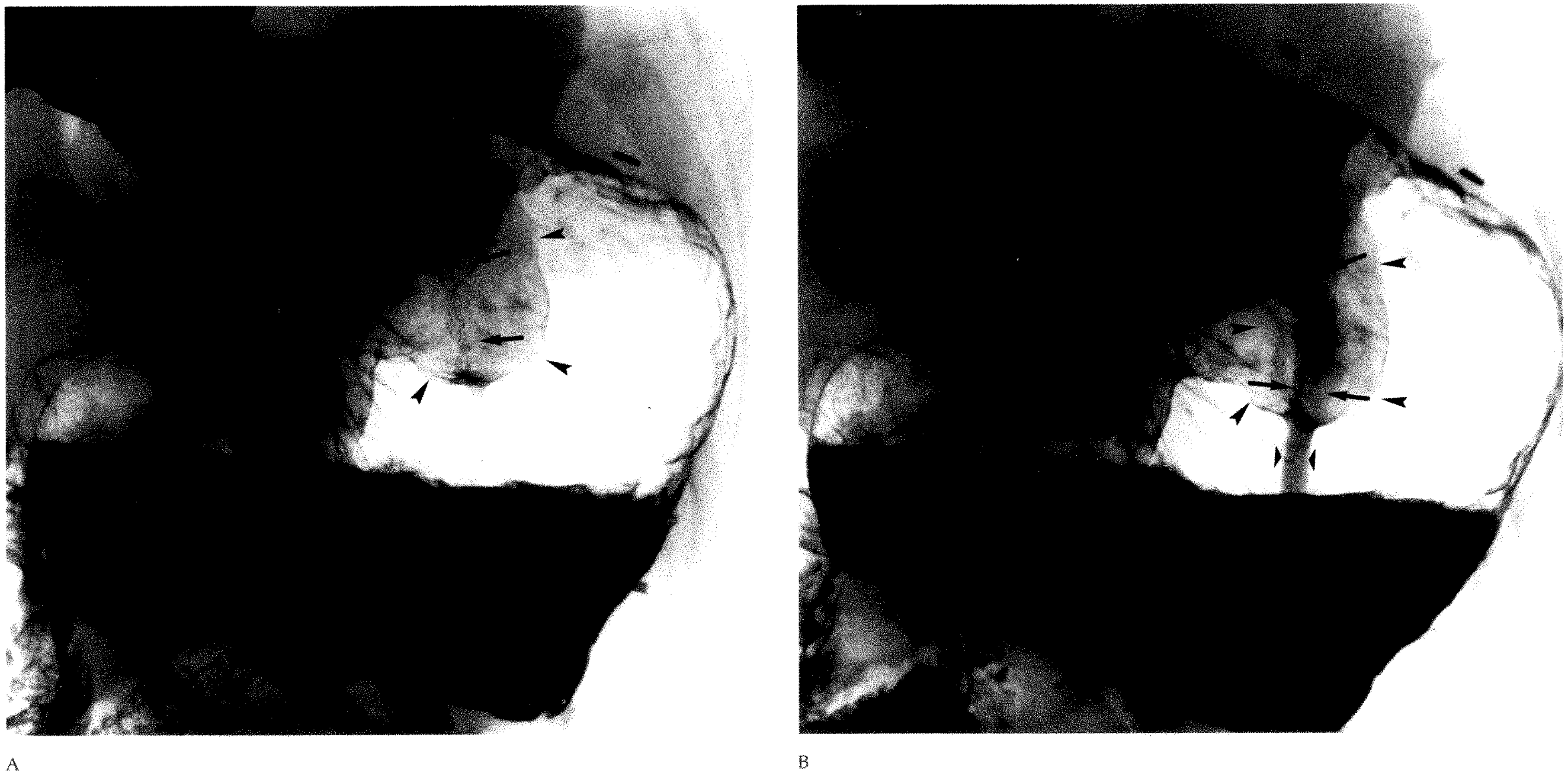

Fig 5. Emptying of barium contrast medium into the stomach. (A) Channel (→) through the antireflux valve (►) closed. (B) Channel through the antireflux valve open while a portion of barium contrast medium (▶ ◀) is emptied from the interposed colon into the stomach.

The channel through the valve seemed to open in response to peristaltic movements in the colon transplant (Fig 5).

The competence of the valve was tested during straining with the patient in the deep Trendelenburg's position. No reflux from the stomach to the colon transplant was seen.

The results of scintigraphy and manometric studies will be presented in a separate report.

Comment

Extensive clinical work has shown that the esophagus can be replaced effectively with a colon substitute [7–13]. Although the stomach is the organ most frequently used, interposition of colon is a valid alternative for a wide variety of benign lesions, particularly in patients with a long life expectancy. Other indications for colon substitution of the esophagus include high primary cancers of the esophagus and hypopharynx necessitating total esophagectomy, esophageal cancers with extension to the stomach, secondary high involvement of the esophagus by thyroid or laryngeal cancers, for example, or unavailability of the stomach because of disease or a previous gastric operation.

Thirty patients have undergone colon replacement of the esophagus at our institution during the last 10 years. The operative mortality rate was 3.3%.

Major and minor complications of colon interposition have been discussed by Gunning and Marshall [5], by Postlethwait in an excellent collective review [14], and by Kotsis and associates [4]. Necrosis of the interposed segment, leakage, and stricture of the proximal anastomosis are the most frequent major complications. Sacculation of redundant interposed colon with prolonged emptying remains a problem with the conventional surgical technique and can necessitate a second operation.

Reflux from the stomach into a colon transplant or into the esophageal remnant can result in inflammation, which can progress to ulceration and stricture. This complication and pulmonary complications from aspiration [5] are the most common in esophagogastrostomy even with high cervical anastomoses [15]. With the conventional techniques for cologastric anastomosis on the posterior or anterior aspect of the stomach, reflux rarely occurs. However, serious complications due to gastrocolic reflux have been reported [2–4, 16]. One of our patients had had several severe episodes of aspiration and bleeding from an anastomotic ulcer. The seriousness of anastomotic ulcers and the difficulty of treating them highlight the need for their prevention [3].

With the method presented here, reflux can be eliminated. The procedure is simple and appears to be safe. An end-to-end cologastric anastomosis with a competent valvular mechanism is achieved that functionally resembles the normal esophagogastric junction. A separate end-to-end cologastric anastomosis is not necessary. The length of the graft is somewhat shorter than with the conventional methods of cologastric anastomosis, thus making the transit of food to the stomach easier. Less risk of redundancy of the graft by adjustment of the length of the valve is another advantage.

Several technical aspects should be emphasized. One is the importance of a gastric drainage procedure, vagotomy, and pyloroplasty, but a feeding jejunostomy probably is unnecessary with increased experience. Another is the use of the left colon, which has more propulsive peristalsis than the right colon. Prolapse of the valve, a well-known complication with the valves used in continent ileostomy reservoirs [17], has not been observed, and fixation of the valve to the gastric wall does not appear to be necessary.

The addition of an intussusception valve at the distal end of an interposed segment of colon prevents gastrocolic reflux without causing any major delay in the passage of food to the stomach. The method offers unique possibilities of studying the function of the interposed colon. The interposed segment of colon empties, at least partially, by means of a propulsive peristalsis.

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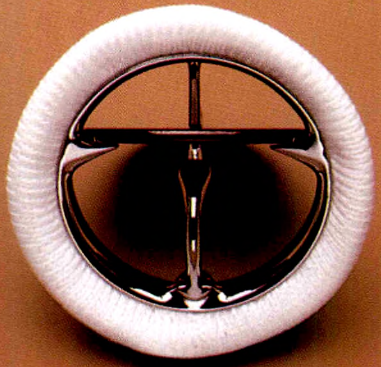

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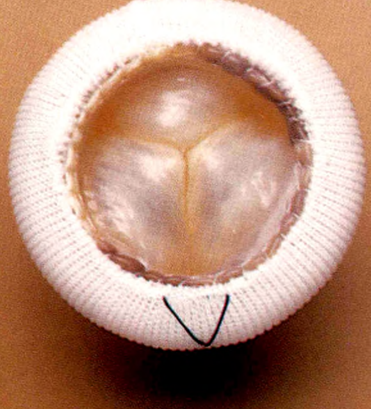

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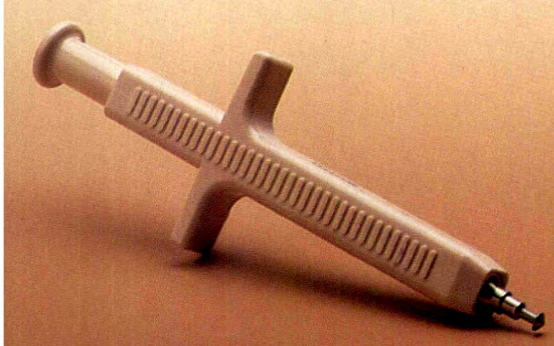

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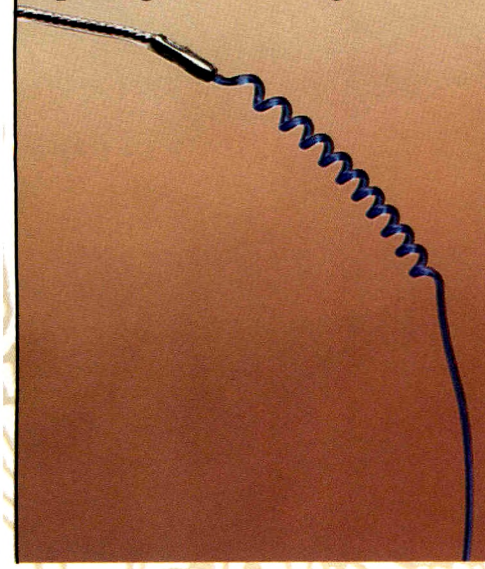

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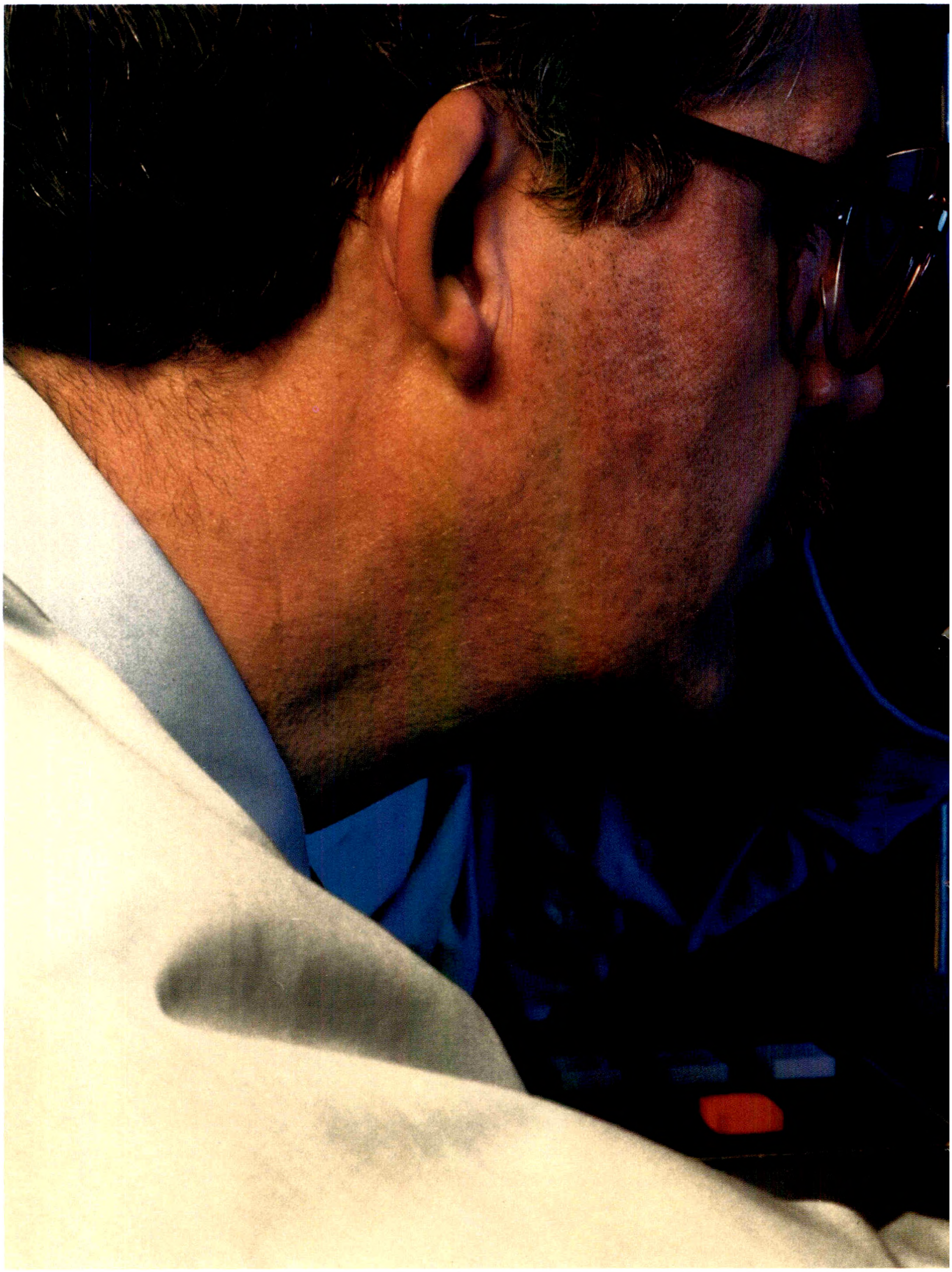

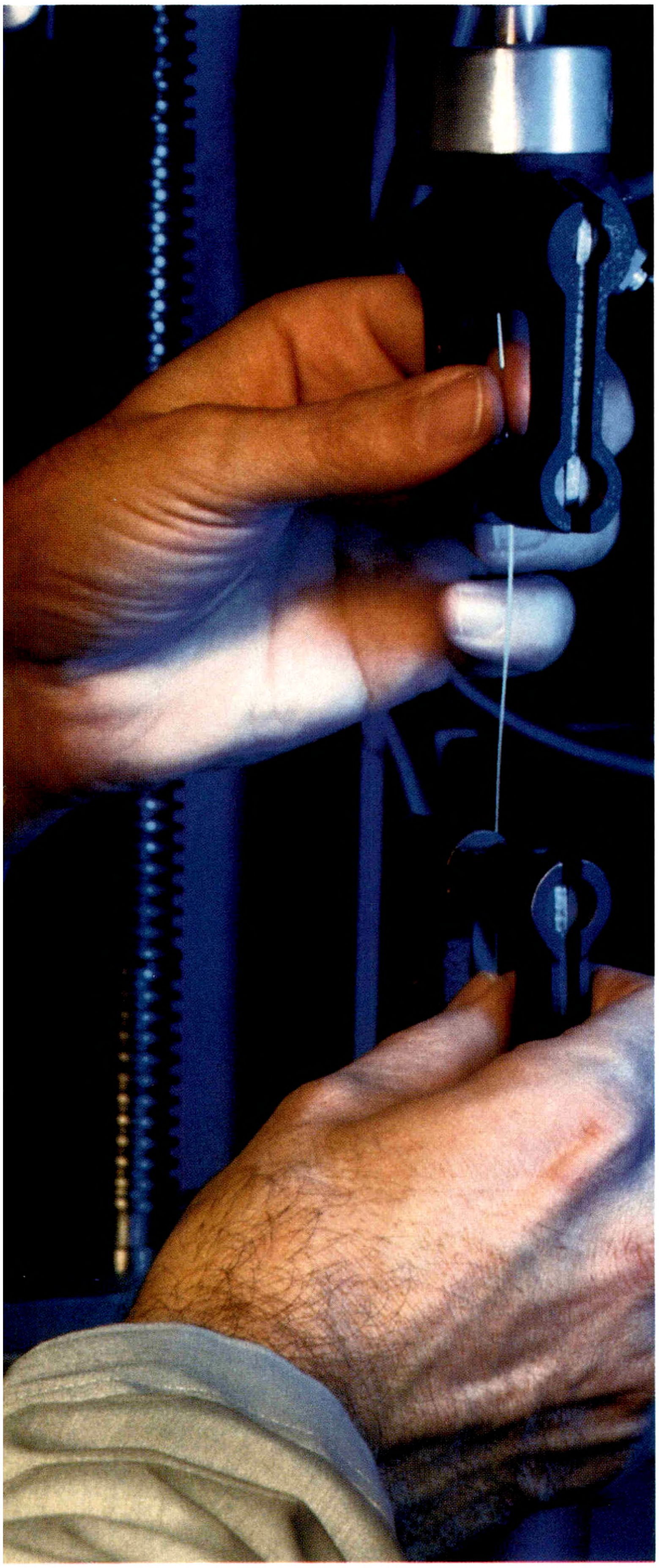

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Tissue Adhesive in Bronchial Closure

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Department of Thoracic Surgery, Bradford Royal Infirmary, Bradford, England

Monomeric *n*-butyl-2-cyanoacrylate was used in 25 patients undergoing pulmonary resections to strengthen the bronchial stump after pneumonectomy (n = 11) and to aid bronchial (n = 13) and tracheal anastomosis (n = 1) after sleeve resections. Neither group had any incidence of bronchopleural fistula. Bronchial anastomosis was accomplished in patients who underwent sleeve resection, reducing the number of sutures required to four apposing sutures, with the tissue adhesive ensuring an airtight closure. There was no incidence of bronchial stenosis. The efficacy of *n*-butyl-2-cyanoacrylate in preventing fistula formation after bronchial resections makes it an ideal agent in pulmonary surgery. Its use obviated the use of pedicled pleural flap, thus ensuring pleural integrity for extrapleural continuous intercostal nerve blockade for postoperative analgesia.

(Ann Thorac Surg 1989;48:683–5)

Despite advances in chemotherapy and radiotherapy, pulmonary resection remains the current preferred treatment for localized non-oat cell carcinoma of the bronchus [1]. In many cases, pneumonectomy must be performed to achieve adequate tumor clearance. In recent years, lung conservation accomplished by using sleeve resections of proximal bronchi has become increasingly popular [1, 2]. The long-term results achieved have been comparable with those achieved by pneumonectomy, with better preservation of pulmonary functions [1].

Bronchopleural fistula remains a dreaded complication of pulmonary resections. Its incidence has decreased in recent years with better understanding of bronchial healing [3] and refinement of surgical techniques. Pneumonectomy still has a 7% rate of bronchopleural fistula [1, 4], resulting in multiple surgical procedures, prolonged hospital stay, and mortality of up to 20% [4]. The corresponding rate for sleeve resection is up to 11% [1]. Sleeve lobectomy with bronchial anastomosis also carries a risk of bronchial stenosis, which has been reported to affect 17% to 20% of patients [2]. To avoid these complications of pneumonectomy and sleeve lobectomy, we have been using monomeric *n*-butyl-2-cyanoacrylate tissue adhesive in patients undergoing such procedures. This report summarizes our recent experience with this technique.

Patients and Methods

Twenty-five patients with T3 tumors underwent pulmonary resections for primary bronchial carcinomas that were within the main bronchus less than 2 cm from the carina without involving it. All patients had full preoperative assessment, including radiography, respiratory function tests, bronchoscopy and biopsy, and mediastinoscopy, and the tumors were considered resectable on these grounds.

Table 1 summarizes data of the patients and the types of resections performed. To obtain maximal tumor clearance, the patients who underwent pneumonectomy were not considered suitable for stapled bronchial closure due to the proximity of the tumors to the carina. After removal of the lungs, the bronchial stump was closed with four equally spaced interrupted polypropylene sutures, and the stump was strengthened with 0.5 g of monomeric *n*-butyl-2-cyanoacrylate (Histoacryl blue, B. Braun, FRG). Airtight closure was achieved in a minute when the adhesive dried. Testing to an inflation pressure of 50 cm H_2O revealed no air leakage.

Fourteen patients underwent sleeve lobectomy. Of these, 13 underwent right upper lobectomy with sleeve resection of the right main bronchus and 1 patient had a sleeve resection of the trachea. Anastomosis was started with four interrupted polypropylene sutures equally spaced around the circumference of the airway to appose the edges, with the knots tied externally. This was completed with monomeric *n*-butyl-2-cyanoacrylate tissue adhesive applied along the entire external circumference of the anastomosis commencing posteriorly. Airtight closure was achieved when the adhesive dried, within a minute of application. Pedicled pleural flaps were not used to strengthen bronchial closure so that an intact pleura could be preserved for extrapleural infusion of bupivacaine for postoperative analgesia. Postoperative bronchoscopy at 6 weeks was routinely performed.

Histological examination of the specimens resected showed 18 squamous cell carcinomas, three adenocarcinomas, one carcinosarcoma, one oat cell carcinoma, one alveolar cell carcinoma, and one large cell carcinoma.

Results

Follow-up was complete in all patients, with a mean of 6 months (range, 1 to 18 months). There was no operative mortality. All patients made an uncomplicated postoperative recovery and were discharged seven to ten days postoperatively. Clinical and bronchoscopic examinations showed no bronchopleural fistula and no evidence of anastomotic strictures.

Accepted for publication July 31, 1989.

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Table 1. Pulmonary Resection in 25 Patients

Procedure	No. of Patients	Mean Age (yr)[a]	Sex	
			M	F
Pneumonectomy				
Left	8	62.5 (52–75)	6	2
Right	3	57.3 (54–62)	2	1
Sleeve resection				
Right upper lobe	13	67.2 (61–73)	13	
Tracheal	1	45	1	
Total	25			

[a] Numbers in parentheses are ranges.

Comment

Bronchial resection and anastomosis carry a substantial risk of fistula formation. Advances in surgical techniques, including use of stapling devices [3], have minimized but not abolished such complications. Our practice is to use stapled bronchial closure after pneumonectomy provided that at least 1.5 cm clearance can be obtained from palpable or visible tumor. In patients who underwent pneumonectomy in this series, the proximity of the tumors to the carina made it necessary for part of the tracheal wall to be resected for adequate clearance. In these patients, stapled closure would lead to serious narrowing of the distal trachea. We therefore used interrupted suture apposition together with tissue adhesive.

Many methods have been advocated to encourage healing and prevent fistulization, including circumferential pleural flap, dermal grafts, intercostal muscle bundle, omentum, pedicled pericardial flaps, and regular postoperative bronchoscopic aspiration of the bronchial trees. These have reduced the incidence of bronchopleural fistula in some series to less than 3% [5]. The absence of any fistula formation in this series has been encouraging.

Airtight closure of the bronchial stump and anastomosis by sutures placed too closely together devascularizes the bronchial tree, leading to fistula formation and stenosis. In addition, sutures tend to cut through the membranous portion of the bronchus, leading to persistent air leak [3]. Wrapping the stump or anastomosis with viable tissues encourages healing but may lead to stenosis of the anastomosis. Using pedicled pleural flaps breaches the pleura, which may usefully be preserved for insertion of an extrapleural catheter for effective postoperative analgesia by continuous infusion of bupivacaine.

Nonsuture anastomosis was studied extensively in the 1960s in arterial [6] and gastrointestinal surgery [7] in experimental animals. The cyanoacrylate group of tissue adhesives was most widely used. These adhesives have been used successfully in preventing hemorrhage from cerebral aneurysm [8] and hemostasis of the heart and major vessels [9]. Sealing of pancreatic resection margin [10] and pancreatic duct obstruction [11] has also been achieved. Toxicity studies of these compounds showed that methyl-2-cyanoacrylate was toxic to the central nervous system in animals [12]. Longer chained monomers, including *n*-butyl-2-cyanoacrylate, have been found to be less toxic [13]. Methyl-2-cyanoacrylate has also been shown to be both bacteriostatic and bactericidal [14] and is slowly degraded and excreted in urine and feces [15].

In experimental animals, monomeric *n*-butyl-2-cyanoacrylate was effective in achieving airtight closure of bronchial anastomosis [16] and proved not to inhibit bronchial healing [17]. Tissue adhesives have been applied in successful management of bronchopleural fistula [18] and esophagobronchial fistula [19]. The ability of the cyanoacrylates to achieve airtight closure in these applications is of significance, coupled with their bacteriostatic and bactericidal properties [14].

Applying monomeric *n*-butyl-2-cyanoacrylate tissue adhesive is a simple and time-saving method for achieving airtight closure in bronchial anastomosis without interfering with healing. This has been demonstrated in experimental animals [17]. Our experience in using this adhesive in pneumonectomy and sleeve lobectomy confirms such experimental findings. By reducing the number of sutures required for bronchial anastomosis, the adhesive may be valuable in preventing bronchial stenosis. Our observation of the lack of toxicity and side effects of this adhesive has been encouraging and was previously confirmed clinically with a 4-year follow-up [20].

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Thromboembolism From Undersized Dacron Grafts in the Descending Thoracic Aorta

Alain Verdant, MD, FRCS(C), Arthur Pagé, MD, FRCS(C), and
Richard Baillot, MD, FRCS(C)

Division of Cardiovascular and Thoracic Surgery, Hôpital du Sacré-Coeur de Montréal, Université de Montréal, Montréal, Québec, Canada

Three patients were referred to our institution for major thromboembolic complications secondary to the use of undersized Dacron grafts (14, 16, and 18 mm) in the descending thoracic aorta. The progressive accumulation of thrombotic material in the prosthesis caused recurrent coarctation in 1 patient and peripheral embolisms in the other 2. With a 9-mm Gott shunt providing distal perfusion, excision of the clotted graft and its replacement with a 22-mm Dacron prosthesis was successfully achieved in each patient.

(Ann Thorac Surg 1989;48:686–8)

Dacron graft interposition is a standard procedure used to reestablish aortic continuity during repair of congenital, traumatic, or degenerative lesions of the descending thoracic aorta. Implantation of an appropriate graft may be difficult at times because the precise diameter of a clamped aorta is difficult to appreciate. We report 3 patients referred to us because the previous insertion of undersized Dacron conduits induced thromboembolic complications that necessitated their replacement.

Material and Methods

Case Reports

PATIENT 1. A 38-year-old man suffered acute arterial occlusion of the left leg 2 weeks after the sudden occurrence of severe claudication of the opposite leg. He had undergone resection of a chronic traumatic aneurysm of the descending thoracic aorta 4 years previously in another institution. In the operative record, it was mentioned that the aortic continuity was reestablished with the interposition of a 16-mm woven Dacron prosthesis. The electrocardiogram was normal. Complete thoracic and abdominal aortography and bilateral lower limb arteriography showed that a tubular aortic prosthesis implanted distal to the left subclavian artery appeared irregular in configuration with a smaller diameter than the native aorta (Fig 1). Embolic material was identified at the bifurcation of the left common femoral artery (Fig 2) and in the right deep femoral and popliteal arteries (Fig 3).

Bilateral femoral embolectomy was undertaken and allowed complete restoration of circulation in both legs. Postoperatively, a pressure gradient of 15 mm Hg between the upper and the lower limbs was demonstrated with plethysmographic studies. One week later, through a left posterolateral thoracotomy, a partially thrombosed 16-mm Dacron graft was removed and easily replaced by a 22-mm woven prosthesis. Perioperative aortic pressure measurements ruled out residual gradient across the aortic repair.

PATIENT 2. A 35-year-old man was investigated for the recent development of severe claudication of the right calf. Three years previously, a left thoracotomy had been performed in another institution for the resection of a 14-year-old posttraumatic aneurysm of the proximal descending aorta. At plethysmography, a gradient of 20 mm Hg between the upper and lower limbs was found. Thoracic aortography and lower limb arteriography showed a filling defect due to the presence of a thrombus in the previously inserted graft in the descending thoracic aorta and the presence of an embolus at the bifurcation of the right popliteal artery.

The old graft, which measured 18 mm, was removed and replaced by a 22-mm woven prosthesis. No further correction was needed for the right popliteal artery stenosis because the complete release of the proximal aortic gradient provided a substantial improvement of the right-leg claudication.

PATIENT 3. A 40-year-old patient underwent surgical correction of an isthmic coarctation 15 years previously in another hospital. A long resection was needed and a 14-mm Dacron prosthesis was used to reestablish aortic continuity. He was referred to us for the reappearance of severe hypertension (190/100 mm Hg). A gradient of 40 mm Hg measured between the upper and lower limbs confirmed the recurrence of coarctation syndrome.

Aortography showed the prosthesis to be very irregular and occupied by a huge thrombus, occluding its lumen almost completely. The removal of the clotted Dacron prosthesis was needed, and its replacement was achieved with a 22-mm woven Dacron graft.

Postoperative plethysmographic studies confirmed the reestablishment of a normal arterial pressure (130/80 mm Hg) and the complete disappearance of the gradient between the upper and lower limbs.

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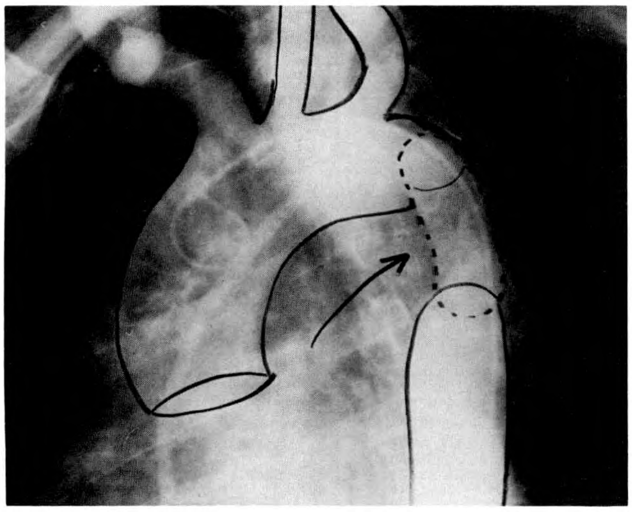

Fig 1. The arrow *indicates irregularities in a small Dacron prosthesis implanted in the aortic isthmus.*

Operative Technique

In the 3 patients, the lesion was approached through a left posterolateral thoracotomy. Organ protection was assured with a 9-mm Gott aneurysm shunt inserted proximally in the ascending aorta and distally in the descending aorta, representing our standard technique of external shunting previously described with details [1].

Operative Findings

Operative findings were identical in the 3 patients. At the opening of the aortic prosthesis, a thick and irregular layer of thrombotic material was found, which was distributed circumferentially, partially occluding its lumen. No false aneurysm and no kink could be identified. The single anatomical defect found as a predisposing factor to clot formation was an obvious discrepancy between an undersized aortic graft and the native aorta.

Results

The 3 patients survived with preservation of the normal function of the spinal cord and other surrounding organs.

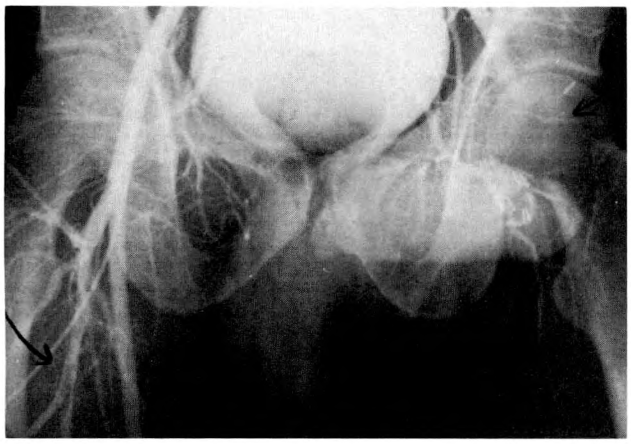

Fig 2. Right deep femoral artery and left common femoral artery embolisms (arrows).

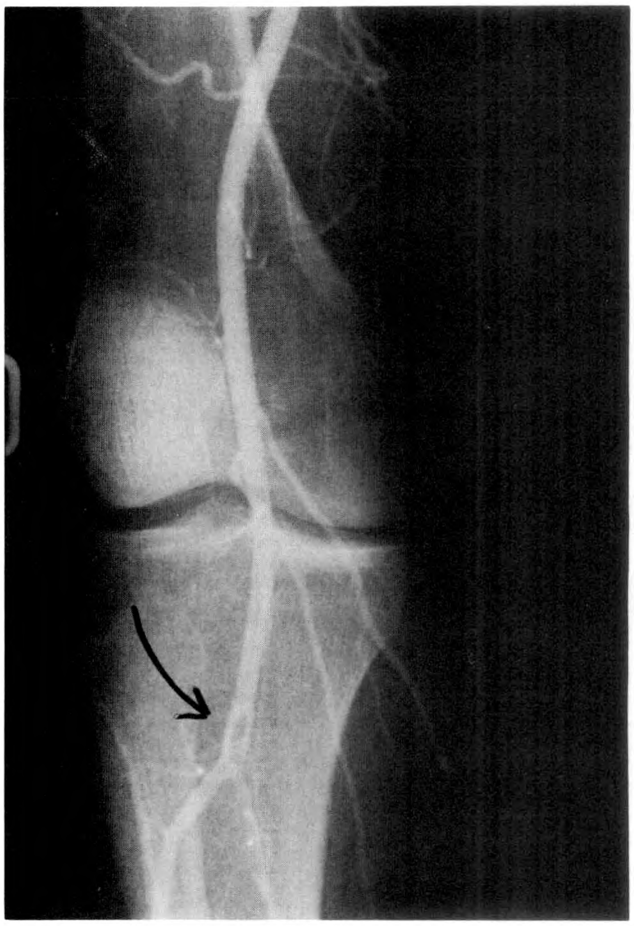

Fig 3. Embolic material in the right popliteal artery (arrow).

Postoperative Doppler and plethysmographic studies confirmed restoration of normal aortic circulation at rest and at exercise without gradient across the new aortic repair.

Comment

Spontaneous detachment of the thrombotic material contained in a descending thoracic aortic aneurysm may occur. Recently, we have successfully resected an 8-cm arteriosclerotic aneurysm of the proximal descending aorta, responsible for an acute mesenteric embolism that necessitated a segmental small bowel resection 3 months before the thoracic aortic procedure.

Other authors have also pointed out lesions of the descending thoracic aorta as a primary source of distal embolization [2–4]. Stallone [2] reported the case of a 41-year-old man involved in a major car accident, who was brought to the emergency room with an acute occlusion of the left popliteal artery. At exploration, the artery had no evidence of trauma. As the patient became hypertensive and the hematocrit dropped without any obvious reason, a thoracic aortogram was performed some 24 hours after the accident. This led to the diagnosis of an unsuspected transected aorta as a source of the popliteal embolism.

Roon and Sauvage [3] have described a right lower limb "blue toe" syndrome in a young patient 6 weeks after a major motor vehicle accident. Thoracic angiography demonstrated a false aneurysm of the descending thoracic aorta not suspected on the day of the accident, and responsible for the embolic event.

Prosthetic grafts inserted with anatomical distortion may also induce thromboembolic complications. Stratton and Hall [4] have reported a pseudointimal embolism from a woven Dacron graft implanted 4 years previously in the descending thoracic aorta for the repair of an acute aortic transection. The indication for reoperation was recurrent embolic phenomena during a 1-month period, angiographically located in the left superficial femoral artery, both renal arteries, and the splenic artery. The size of the graft used is not mentioned. Preoperative thoracic aortography disclosed some evidence of redundancy and a slight kinking of the graft, which was confirmed at thoracotomy. An area of denuded pseudointima was also observed, suggesting clearly that the embolic process was originating at this precise site. Stratton and Hall postulated that the embolic phenomena were related to inadequate adherence of the pseudointima to the woven fabric, and recommended repairing descending thoracic aortic aneurysms with knitted prostheses.

From our experience with more than 215 cases of resection of descending thoracic aortic aneurysms and replacement with woven prostheses, there was no distal embolization from the aortic graft. Consequently, we cannot share the opinion expressed by Stratton and Hall about woven prostheses. In our opinion, woven grafts are reliable and have little propensity to dilate in time, compared with knitted grafts [5].

In the thoracic aorta, the flow is so rapid that neoendothelialization is limited to a fine lining, unless a gross anatomical defect causing turbulence disturbs this normal healing process. A close examination of the 3 cases reported by us, and the 3 others quoted, shows three types of gross anatomical distortion of the aorta as a source of clot formation: (1) a posttraumatic false aneurysm [2, 3], (2) a kinked synthetic graft [4], and (3) a graft smaller than the native aorta with a substantial gradient across the aortic repair (present series).

The case reported by Stratton and Hall [4] emphasizes the need for a perfect surgical technique to avoid any bulging or kinking of the graft. The lesson to draw from our 3 cases is that a judicious choice of the size of the prosthetic graft is mandatory, so that the final reconstruction reflects as close as possible the diameter of the native aorta.

The average diameter of the thoracic aorta distal to the left subclavian artery varies from 20 mm to 30 mm in young adults. However, when the thoracic aorta is clamped, the aortic segment between the aortic clamps collapses and its real diameter may easily be underestimated.

Coarctation of the aorta and acute traumatic rupture of the descending aorta are two conditions in which an apparently small aorta may be encountered, particularly in the aortic segment distal to the lesion. A complete correction of the coarcted segment is of the utmost importance for relieving the hypertension. When a prosthetic graft has to be used to replace the aorta, an incision extended either on the aortic arch or on the left subclavian artery may have to be performed to increase the diameter of the proximal anastomosis and permit the implantation of at least a 20-mm prosthesis.

In acute traumatic rupture of the descending aorta, we have frequently encountered extremely small proximal and distal retracted aortic ends when the aorta has been torn circumferentially. To overcome this difficulty, we usually incise obliquely the proximal and distal anastomotic sites, and also dilate gently the aorta with a clamp to relieve the spasm at both transected extremities. This usually allows the insertion of a prosthetic graft with a larger diameter than previously expected, which will adapt more precisely to the native aorta.

In a recent editorial by Barner [6], the use of a sutureless intraluminal aortic prosthesis in traumatic aortic rupture is highly questioned, and we agree with his argument. We also disapprove of the use of this device in young adults for the following reason not mentioned in the editorial. The effective luminal diameter of the graft is 4 to 5 mm smaller than the external diameter of the fixation ring. Consequently, to accommodate any basic aortic diameter, the Dacron graft used with this method will have a smaller size than the one obtained with a conventional Dacron graft with suture anastomosis. This may result in a stenotic repair with the further development of thromboembolic complications.

Conclusion

The constant threat of paraplegia in descending thoracic aortic surgery puts some stress on the surgeon, usually very anxious to expedite the procedure with the shortest aortic cross-clamping time feasible. The rapidity of execution of the aortic replacement should not prejudice the surgeon to a precise technique, because an inadequate reconstruction may induce further thromboembolic complications. The choice of the optimal size and length of the prosthetic graft will avoid redo procedures, which may entail a much higher morbidity for the patient. To accommodate the size of an adult aorta, we consider a 20-mm diameter as the smallest acceptable size for a Dacron conduit to reestablish aortic continuity at the isthmus.

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Replacement of the Descending Thoracic Aorta With Intraluminal Ring Graft

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Renal failure and paraplegia are major complications of operations on the descending thoracic aorta. To minimize cross-clamp time and reduce the incidence of such complications, we have used an intraluminal ring graft to reestablish aortic continuity in patients with descending thoracic aortic lesions. From March 1978 to December 1986, we used this technique alone in 28 patients. There were 4 patients with traumatic aortic disruptions, 4 with dissections, 4 with expanding aneurysms, 2 with ruptured aneurysms, 1 with Marfan's syndrome, and 13 with atherosclerotic aneurysms that were repaired electively. The cross-clamp times ranged from 4 to 28 minutes. There were three early postoperative deaths (within 30 days) and one late postoperative death in the follow-up period (mean, 28.2 months). Ring dislodgement occurred only once, in the first patient in this series. There were no instances of postoperative renal failure or paraplegia. We conclude that the use of an intraluminal ring graft greatly reduces the aortic cross-clamp time and is a safe and effective technique.

(Ann Thorac Surg 1989;48:689–92)

Aortic cross-clamping, which is necessary in operations on aneurysms or dissections of the descending thoracic aorta, has been associated with complications related to the distal ischemia produced—the most frequent and feared of which is paraplegia. Several methods have been proposed to avoid these complications [1–5], but none has eliminated the incidence of paraplegia, which seems to have a multifactorial cause [6].

Because the incidence of paraplegia increases with periods of cross-clamping longer than 20 minutes [7], it would seem that the ideal surgical technique for replacement of the descending thoracic aorta should be simple and rapid. We previously described our experience with the use of an intraluminal ring graft in the replacement of thoracic and abdominal aortic segments [8]. Between 1976 and 1978, patients who underwent descending thoracic aortic repair were supported with femorofemoral bypass. Since 1978, simple cross-clamping without cardiopulmonary bypass has been used exclusively.

The purpose of this report is to emphasize the feasibility of replacing the descending thoracic aorta with a short cross-clamp time and minimal morbidity.

Material and Methods

Patients

From March 1978 to December 1986, 37 patients with descending thoracic aortic lesions were operated on without cardiopulmonary support. We were able to use both rings of the intraluminal graft in 28 patients, and these patients are the subjects of this report. There were 21 men, with ages ranging from 15 to 73 years (mean age, 56 years; standard deviation [SD], 16), and 7 women, with ages ranging from 45 to 71 years (mean age, 62.83 years; SD, 10.30). There were 4 patients with traumatic aortic interruptions, 4 with acute dissections, 4 with expanding and 2 with ruptured aneurysms, 1 patient with Marfan's syndrome, and 13 patients with atherosclerotic aneurysms to be repaired electively. All of the dissections started distal to the left subclavian artery and were confined to the descending thoracic aorta, except in 1 patient in whom it extended to the upper abdominal aorta. The cross-clamp times ranged from 4 to 28 minutes (mean cross-clamp time, 9.5 minutes; SD, 6.5).

Over the last 3 years, a digital subtraction arteriogram has been performed in all patients before discharge. Follow-up ranged from 2.5 to 48.5 months (mean follow-up, 28.23 months; SD, 17.18).

Operative Technique

A posterolateral left thoracotomy is used in all patients. The pleural cavity is entered through the third, fourth, fifth, or sixth interspace, depending on the location of the aneurysm or dissection. Removal of the rib is unnecessary, although disarticulation is sometimes helpful. Occasionally, in very large and long aneurysms, incisions through two different intercostal spaces are necessary. After the thorax has been entered and the lung has been retracted away from the aneurysm or dissection, immediate control of the aorta above and below the lesion is accomplished by encircling the aorta with a nylon tie, avoiding touching the aneurysm or dissection. The aorta then is mobilized further for cross-clamping proximally and distally. The takeoff of the large vessels is evaluated to determine if the aorta can be occluded distal to the subclavian artery to allow insertion of the rigid spool. If this cannot be done, the aorta is prepared between the left carotid and subclavian arteries so that it can be cross-

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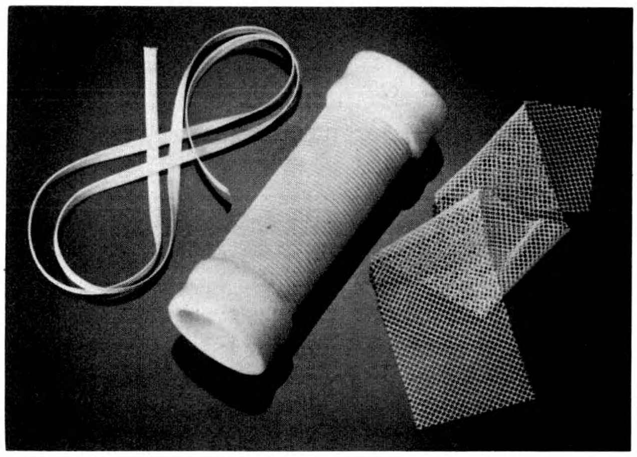

Fig 1. Intraluminal ring graft.

clamped at that point. Next, 5,000 U of heparin are injected intravenously. The appropriate sized graft is chosen by discounting 30% (for radiologic augmentation) from the aortic diameter measured on the arteriogram.

The graft (Fig 1) is autoclaved in 5% albumin for three minutes. The aorta then is cross-clamped, and a longitudinal incision is made through the involved area. Intravascular clots are removed, and in the case of a dissection, the tear is identified. For thoracic aneurysms, the limiting borders are determined, and the graft is inserted so that the spools are placed proximally and distally. To accomplish this, a longer graft often is needed. This is created by using two ring grafts with a Gore-Tex graft of appropriate diameter (usually the same as the size of the spools) interposed between them and tied to the rings on each side (Fig 2). The size of the Gore-Tex graft is tailored according to the length necessary to replace the aneurysm completely. Telescoping the Gore-Tex graft over the rings allows for further modification of the prosthesis length (Fig 2).

In the case of a dissection, the lumen is identified and the proximal spool of the graft is always placed above the entrance site. With the graft well placed and tied, the tear is excluded from the blood stream and the false lumen is effectively occluded. Double-tying over the rings approximates the flaps appropriately (Fig 3).

Intercostal artery back-bleeding is assessed qualitatively. If the back-bleeding is minimal (usually in the case of aneurysm, the intercostal arteries are occluded by clots), nothing is done; if it is brisk, the intercostal arteries are oversewn.

With the surgeon holding the proximal spool in place so that it is axially aligned with the aorta, the assistant ties two nylon tapes around the outside of the aorta in the groove of the spool, securing it in place. At this point, the lumen is suctioned and inspected for any debris dislodged during fixation of the proximal spool, and the distal spool is inserted and tied into the distal aorta using a similar technique. The proximal aorta then is opened briefly to allow air in the graft to escape through the interstices. The distal aorta then is opened completely, and the operative site is checked for any major leaks. At this point, there will be some bleeding through the graft interstices, but this is usually stopped with administration of protamine and compression of the wall of the aorta. The proximal cross-clamp is opened slowly while adequate blood pressure is maintained, and the site is inspected for excessive bleeding. More recently, to avoid the distal aortic cross-clamp, we have introduced the distal spool first over a 30-mL Foley catheter that controls the aorta when inflated. With this technique the proximal spool is inserted last. Two strips of Teflon felt, approximately 1 inch wide, are cut to the length of the aortotomy. The longitudinal incision of the aorta then is closed using horizontal mattress sutures of 3-0 Ethibond through the Teflon felt sandwich. A second layer of vertical mattress sutures is placed for hemostasis. The distal aorta and the vessels of the arch are inspected for good pulsatile flow. Heparin is reversed with intravenous protamine on a 1:1 basis. Chest tubes are inserted and secured. The thoracotomy incision then is closed in the classic fashion.

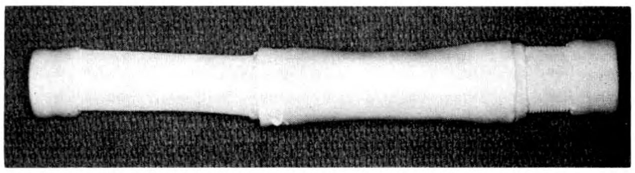

Fig 2. Gore-Tex graft interposed (being "telescoped") between two ring grafts.

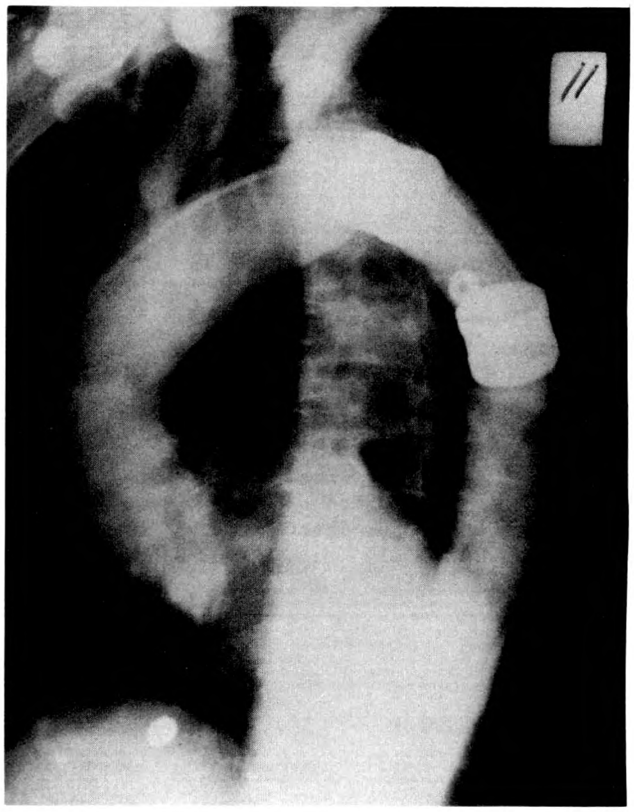

Fig 3. Postoperative arteriogram of a type IIIa aortic dissection.

Results

There were three (10.7%) early postoperative deaths (within 30 days) and one (3.6%) late postoperative death in the follow-up period. The early postoperative deaths were in a 61-year-old patient with a ruptured descending thoracic aortic aneurysm who had postoperative bleeding requiring exploration and died on the first postoperative day; a 67-year-old patient who underwent resection of a descending thoracic aortic aneurysm and died in the immediate postoperative period; and a 69-year-old patient who underwent resection of a descending thoracic aortic aneurysm and had a postoperative course complicated by bronchopneumonia requiring prolonged ventilatory support and small bowel resection. At autopsy, graft placement was intact in all patients. The late death was the first patient of the series. This 32-year-old man suffered later dislodgement of the graft rings requiring reoperation and died 6 months after the initial procedure. There was one instance of postoperative gastrointestinal bleeding due to a stress ulcer, which was controlled adequately with medical therapy. No cases of postoperative renal failure or paraplegia were documented. There were no late complications associated with thrombosis, erosion, or pseudoaneurysm.

Comment

The modern surgical approach to the thoracic aorta was heralded by DeBakey and associates [9] in their report on operations for dissecting aortic aneurysms.

Following this report, an aggressive surgical approach was used by others with success. After the initial experience with a fenestration procedure, DeBakey and colleagues [10] rapidly switched to aneurysm resection and graft replacement, which is today's accepted technique. With experience, better surgical timing [11–15], improved operative and postoperative care, and refinement of surgical techniques have resulted in better results [11].

Our operative mortality rate seems to be slightly lower than that in some of the reports of large series [6, 10]; this may be partially related to improvement in operative technique and postoperative care [10, 11]. Most of the recent interest in this field has been focused on the attempt to diminish the incidence of complications related to the distal organ ischemia, and much emphasis has been placed on the need for a simplified and rapid surgical technique that would prevent such complications [6].

Anatomical variations of the spinal cord arterial supply are difficult to assess intraoperatively [6].

We feel that one of the primary causative factors of postoperative paraplegia that can be controlled is intraoperative ischemic time. In our series, we were able to perform the operation with a minimal cross-clamp time. In 1 patient, we had an aortic cross-clamp time of four minutes, and in 2 other patients, the cross-clamp time was 4.5 minutes. Once, in a patient in whom the aorta needed to be reclamped for ten minutes for control of blood pressure, the total cross-clamp time was greater than 20 minutes. It is known that the incidence of paraplegia increases with cross-clamp times longer than 20 minutes [7]. Our short distal aortic ischemic time may explain the absence of paraplegia in our patients as well as the absence of renal failure.

In about 24% of the cases, we were not able to use one of the graft spools, usually the proximal one, because of absence of sufficient space distal to the left subclavian artery. In these instances, the spool was removed and the graft sutured. This situation has been occurring less frequently in our experience. In recent cases, when there is not enough space distal to the left subclavian artery, we opt to place the spool distal to the left carotid artery; and, after the aortic cross clamp is removed, we place a 10-mm Gore-Tex graft between the intraluminal prosthesis and the left subclavian artery. The use of the intraluminal ring graft greatly reduces the aortic cross-clamp time and is a safe and easily reproducible technique. Even in cases in which one of the spools has to be removed and the graft handsewn in the usual fashion, the benefit is still substantial because fixation of the spool in the distal end contributes to a shorter cross-clamp time. Very early in our experience there was one instance of a graft dislodgement that required reoperation. Six months subsequent to operation, the patient died of empyema. There were no other cases of graft dislodgement.

To decrease the incidence of late aneurysm formation, we emphasize the importance of blood pressure control in all our patients [11].

We realize that longer follow-up is necessary for proper assessment of the incidence of false aneurysm formation. However, the absence of pseudoaneurysm, erosion, and thrombosis during this initial follow-up is encouraging.

This technique remains our procedure of choice for aortic replacement and is a safe and effective surgical option in the treatment of descending thoracic aortic lesions.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. They should also keep a record of their attendance at thoracic surgical meetings, and other continuing medical education activities, for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS III syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS III booklets prior to applying for recertification. SESATS III booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1992 may begin the recertification process in 1990. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1990. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Clinical Features of Aortic Arch Anomaly With Malalignment Ventricular Septal Defect

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The clinical features and outcome after various surgical procedures on 9 patients with coarctation or interruption of the aortic arch and malalignment ventricular septal defect (group 1) were compared with those of 9 patients with the arch anomaly without malalignment ventricular septal defect (group 2). Cardiomegaly and metabolic acidosis were prominent in group 1. Five of the 9 patients in group 1 died in the immediate postoperative period (56% mortality), but no operative deaths occurred among 8 patients in group 2 ($p < 0.01$). The ratio of left ventricular outflow tract to ascending aortic diameter was 0.59 ± 0.09 in group 1 and 1.03 ± 0.11 in group 2 ($p < 0.01$). Three of 4 patients with a ratio of less than 0.6 died, but no operative deaths occurred among the 6 patients who had a palliative operation and in whom the ratio was more than 0.6. These data suggest that left ventricular outflow tract obstruction is critical when the ratio of left ventricular outflow tract to ascending aortic diameter is 0.6 or less. The presence of severe left ventricular outflow tract obstruction necessitates modification of the present surgical strategy.

(Ann Thorac Surg 1989;48:693–6)

The morphology of ventricular septal defects in hearts with coarctation or interruption of the aorta has previously been described [1–5]. According to these studies, malalignment defects with posterior deviation of the conal septum into the left ventricle result in obstruction of the left ventricular outflow tract and divert ventricular blood flow into the pulmonary trunk and away from the aorta. When the obstruction is severe, the clinical features would be expected to be changed. There is little information about the clinical features and the prognosis of these anomalies associated with malalignment defects.

This report describes the clinical features and the prognosis of coarctation and interruption of the aorta with malalignment defects, and discusses the critical point of severity of left ventricular outflow tract obstruction and its influence on surgical management.

Material and Methods

In the 13-year period from January 1975 through December 1987, 18 patients were seen with a diagnosis of coarctation of the aorta or interruption of the aorta with ventricular septal defect. All these hearts demonstrated visceroatrial situs solitus, atrioventricular concordance, and ventriculoarterial concordance.

The diagnosis of coarctation or interruption of the aorta was confirmed by preoperative echocardiography or cardiac catheterization and angiography in 17 patients, and by postmortem examination in 1.

The diagnosis of malalignment ventricular septal defect was made by echocardiography in the parasternal left ventricular long axis view (Fig 1) or by the left ventriculogram in the left anterior oblique. The precise echocardiographic approach to define the malalignment ventricular septal defect was described by Smallhorn and associates [6].

Nine of 18 patients had malalignment ventricular septal defect (group 1). They constitute the main material for this study. The remaining 9 patients had ventricular septal defect without malaligned conal septum (group 2). Three patients in group 1 and 1 in group 2 had interruption of the aortic arch.

The severity of the left ventricular outflow tract obstruction was echocardiographically assessed by using the ratio of the diameter of the left ventricular outflow tract to that of the ascending aorta (LVOT/AAo ratio). Measurements of the diameters of the left ventricular outflow tract proximal to the aortic valve, and of the ascending aorta distal to the aortic cusp, were made from parasternal long axis view of echocardiogram during systole.

Clinical manifestations, mortality rate, LVOT/AAo ratio, and surgical procedure were evaluated and compared between groups 1 and 2.

Statistical analysis was performed using the Student's t test and χ^2 test. A value of $p < 0.05$ was considered statistically significant.

Results

The LVOT/AAo ratio ranged from 0.50 to 0.71 (mean, 0.59 ± 0.09) in group 1, and from 0.86 to 1.20 (mean, 1.03 ± 0.11) in group 2 ($p < 0.01$). The significantly lower ratio in group 1 indicated that the left ventricular outflow tract

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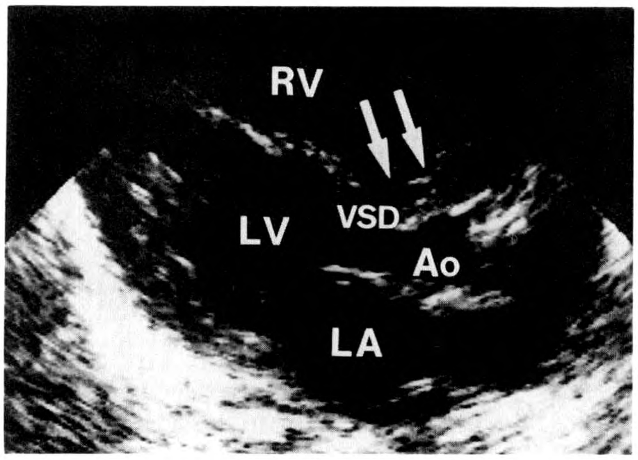

Fig 1. Precordial long axis view in coarctation and malalignment ventricular septal defect (VSD). Note the ventricular septal defect and the conal septum, which is displaced posteriorly into the left ventricular outflow tract (arrows). (Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.)

Table 1. Comparison of Clinical Features Between Groups 1 and 2

Feature	Group 1	Group 2	p Value
Birth weight (g)	2,955 ± 480	3,310 ± 459	NS
Sex (M/F)	3/6	3/6	NS
Onset of CHF (days)	21 ± 27	20 ± 28	NS
Administration of inotropic agents and diuretics	9/9	9/9	NS
Administration of PGE$_1$	6/9	3/9	NS
Blood gas analysis			
pH	7.26 ± 0.14	7.36 ± 0.06	NS
Base excess	−8.7 ± 9.9	−2.0 ± 3.8	NS
Electrocardiogram			
Axis (degrees)	142 ± 64	103 ± 84	NS
ST-T change	8/9	5/9	NS
Chest roentgenogram			
CTR			
Mean (%)	70 ± 4	66 ± 3	<0.05
<70%	6/9	1/9	<0.05
Increased pulmonary blood flow	9/9	9/9	NS
Age at operation (days)	75 ± 68	68 ± 50	NS
Body weight at operation (g)	3,456 ± 850	3,508 ± 727	NS
Period of intubation (days)	37 ± 37	19 ± 15	NS
Operative mortality rate	5/9 (56%)	0/8 (0%)	<0.01

CHF = congestive heart failure; CTR = cardiothoracic ratio; PGE$_1$ = prostaglandin E$_1$.

obstruction was more severe in group 1 than in group 2 (Fig 2).

Five of the 9 patients in group 1 died in the postoperative period (56% mortality). In contrast, there were no operative deaths among 8 patients in group 2 ($p < 0.01$). The last patient in group 2 died of sepsis with acute renal failure preoperatively.

The clinical data are summarized in Table 1. There were no differences between the two groups with respect to birth weight, sex, onset of congestive heart failure, administration of inotropic agents, diuretics, and prostaglandin E$_1$, electrocardiogram, age and body weight at operation, and period of intubation.

Blood gas analysis revealed that mean blood pH and base excess were 7.26 ± 0.14 and −8.7 ± 9.9 in group 1 and 7.36 ± 0.06 and −2.0 ± 3.8 in group 2, respectively. Although there were no statistical differences between the two groups, severe metabolic acidosis was present in 4 of 9 patients in group 1.

Cardiothoracic ratio on chest roentgenogram was significantly higher in group 1 (mean, 70% ± 4%) than in group 2 (mean, 66% ± 3%) ($p < 0.05$).

Figure 3 shows the relation between the severity of left ventricular outflow tract obstruction assessed by the LVOT/AAo ratio and the outcome after various surgical procedures.

Three of 4 patients with severe obstruction of the left ventricular outflow tract (LVOT/AAo ratio less than 0.6) died shortly after operation regardless of the type of operative procedure for aortic arch reconstruction and whether they underwent pulmonary artery banding or not. In contrast, there were no operative deaths among the 6 patients with comparatively mild obstruction of the left ventricular outflow tract (LVOT/AAo ratio greater than 0.6) who underwent palliative operation.

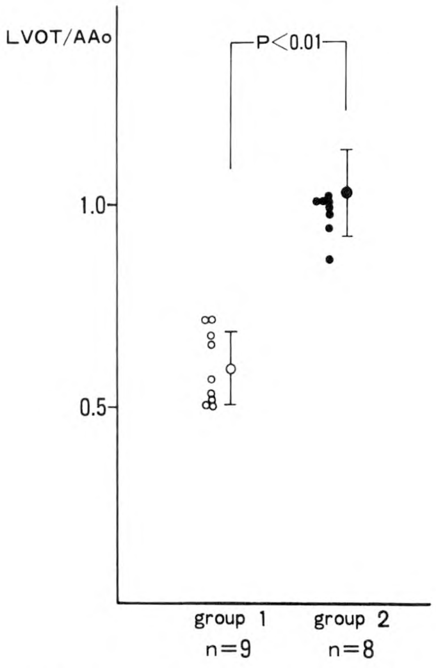

Fig 2. The ratio of the diameter of the left ventricular outflow tract (LVOT) to that of the ascending aorta (AAo).

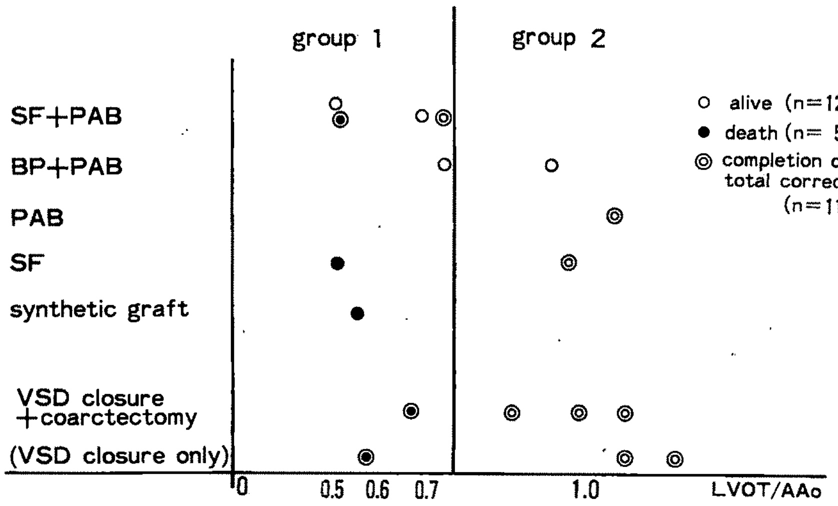

Fig 3. The relationship between LVOT/AAo ratio and outcome after various surgical procedures. (AAo = *ascending aorta;* BP = *Blalock-Park method;* LVOT = *left ventricular outflow tract;* PAB = *pulmonary artery banding;* SF = *subclavian flap method;* VSD = *ventricular septal defect.)*

Four patients underwent closure of the ventricular septal defect at the same time as relief of the coarctation. Three patients underwent closure of the ventricular septal defect only. Among these 7 patients, both of those with an LVOT/AAo ratio of 0.7 or less died.

Comment

Recent reports [5, 6] have addressed the type of ventricular septal defect associated with coarctation of the aorta. Such defects tend to be seen earlier and with more severe cardiac failure. Our experience indicates a significantly higher operative mortality rate for malalignment defects compared with simple perimembranous defects.

Regarding clinical features, cardiomegaly and metabolic acidosis on admission were found to be slightly more common in patients with malalignment ventricular septal defect than in patients without malalignment ventricular septal defect.

The LVOT/AAo ratio, which is an indicator of the severity of left ventricular outflow tract obstruction, correlated strongly with operative mortality.

Our results suggest that the left ventricular outflow tract obstruction is critical when the LVOT/AAo ratio is 0.6 or less for palliative operation and is 0.7 or less for the operation of coarctectomy with simultaneous closure of ventricular septal defect. We therefore recommend the strategy depicted in Figure 4.

In coarctation and interruption of the aorta without malalignment ventricular septal defect, closure of the ventricular septal defect at the same time as coarctectomy can be carried out safely. If the ventricular septal defect is small or moderate, coarctectomy alone may suffice. It is known that about 40% to 50% of ventricular septal defects associated with coarctation can become smaller or can close spontaneously with time [11–15], although we did not have such a case in our study group.

In patients with malalignment ventricular septal defects, the LVOT/AAo ratio should be evaluated. If the ratio is 0.6 or more, the selection of surgical approach should be the same as that in patients without malalignment ventricular septal defect.

Pulmonary artery banding is a subject of some controversy. Stafford and associates [16], Penkoske and colleagues [17], and Kamau and co-workers [13] demonstrated that pulmonary artery banding might in itself increase the mortality and morbidity. Hammon and colleagues [18] and Litwin and associates [19] disagreed with their conclusion.

In our series, pulmonary artery banding with coarctec-

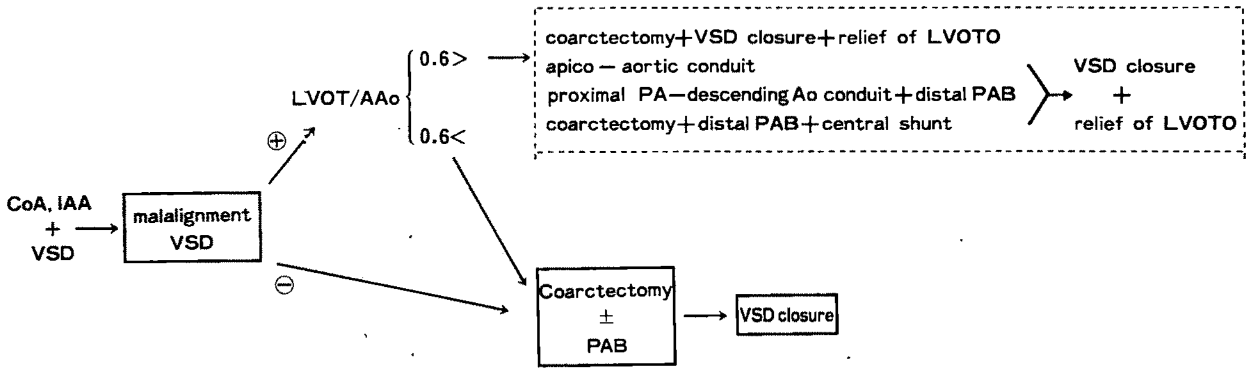

Fig 4. Recommendation for choice of surgical approach in patients with coarctation of the aorta and malalignment ventricular septal defect. (Ao = *aorta;* CoA = *coarctation of the aorta;* IAA = *interruption of the aorta;* LVOTO = *left ventricular outflow tract obstruction;* PA = *pulmonary artery;* PAB = *pulmonary artery banding;* VSD = *ventricular septal defect.)*

tomy resulted in a 17% (1/6) mortality rate, compared with a 67% (2/3) mortality rate in the palliated patients who were not banded. The band, however, may create or aggravate subaortic stenosis, as postulated by Van Praagh and co-workers [1] and Freed and colleagues [20], and confirmed by Trusler and Freedom [21] in 1979. The necessity of pulmonary artery banding in patients with a ratio below 0.6 could not be proved in this study.

If the ratio is 0.6 or less, conventional surgical approaches carry a high risk and, therefore, a variety of new strategies must be considered. Coarctectomy, closure of ventricular septal defect, and resection of the obstructing muscle at the same time is the optimal treatment if reasonable risk can be demonstrated. Apicoaortic conduit may relieve the left ventricular outflow tract obstruction in older children. However, the operative risk is high in the neonate [22].

Recently, Ilbawi and co-workers [10] described a palliative procedure consisting of distal pulmonary artery banding and placement of a pulmonary artery–descending aorta conduit with good survival rate. Yasui and associates [23] also reported 2 neonates with interruption of the aorta associated with severe aortic stenosis palliated using a similar technique in which the proximal pulmonary artery is used as an outflow tract for the left ventricle.

We also used a modified palliative approach in an infant with interruption of the aorta and severe left ventricular outflow tract obstruction. The approach consisted of coarctectomy, distal pulmonary artery banding, and central shunt between the ascending aorta and the main pulmonary trunk. Although the child died of sepsis, this operation could reduce the pulmonary artery pressure and control the congestive heart failure.

We cannot foresee which operation will be the best choice for patients with an LVOT/AAo ratio of 0.6 or less, but we believe the usual surgical approaches are inadequate. The presence of severe left ventricular outflow tract obstruction clearly necessitates modification of present surgical strategies.

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Does Sternal Fracture Increase the Risk for Aortic Rupture?

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We retrospectively reviewed the records of 99 patients who suffered sternal fractures between 1968 and 1987. Patients ranged in age from 5 to 86 years. The most common cause of injury was a motor vehicle accident. The 99 patients were compared with a concurrent series of 2,106 patients with chest injuries and no sternal fractures. Traumatic aortic rupture occurred in 2 of 99 patients with sternal fractures (2%) and in 75 of 2,106 patients without sternal fracture (3.6%). This difference was not statistically significant by the Fisher exact test ($p = 0.326$). We conclude that traumatic aortic rupture does not occur more commonly in patients with sternal fracture when compared with other patients with blunt chest injuries.

(Ann Thorac Surg 1989;48:697–8)

The tremendous force necessary to cause a fracture of the sternum has led clinicians to believe that the presence of a sternal fracture is a harbinger of severe associated injuries [1]. In particular, it has been thought that ruptured thoracic aorta is more likely to occur with a sternal fracture [2–5], and some authors have suggested the presence of sternal fracture is an indication for aortography [2–4]. These ideas continue to be espoused in the current literature [6]. One group of authors [7] emphasizes that the combination of sternal fracture and widened mediastinal shadow on chest roentgenogram is a firm indication for aortography. These seemingly cogent assumptions have never been subjected to scrutiny by statistical analysis in a large series of patients who have suffered blunt chest injuries. The present study retrospectively examined blunt chest injury victims to test the null hypothesis that traumatic aortic rupture (TAR) does not occur more frequently in patients with sternal fractures as compared with blunt chest injury victims without sternal fractures.

Material and Methods

A retrospective review of 2,205 patients who suffered blunt chest injuries between 1968 and 1987 was conducted. These patients had been admitted to the hospital after blunt chest injury and entered in the St. Paul-Ramsey Trauma Registry. The most common causes of the injuries were automobile accidents and falls (Table 1). The patients ranged in age from 5 to 86 years, with the majority of the patients being in the second and third decades of life (Table 2).

The proportion of patients with sternal fracture who incurred TAR was compared with the proportion of blunt chest injury victims without sternal fracture who suffered TAR. The Fisher exact test was used to test statistical significance.

Table 1. Causes of Injury

Cause	No. of Patients With Blunt Chest Injury and Sternal Fracture	No. of Patients With Blunt Chest Injury and No Sternal Fracture
Automobile accident	70	1,219
Fall	12	371
Crush injury	4	65
Pedestrian mishap	3	183
Motorcycle accident	3	190
Other	7	177
Total	99	2,205

Blunt chest injury was defined as the presence of a specific thoracic lesion, such as rib fractures, sternal fractures, hemothoraces, and pulmonary contusions. "Widened mediastinum" in itself was not defined as blunt chest injury. Consequently, only 57 of the 2,205 blunt chest injury patients underwent aortography. During the same time frame, approximately 300 additional thoracic aortograms were performed for other patients with multiple injuries who did not fit the criteria for having suffered blunt chest injury, but who demonstrated "widened mediastinum" on chest film or had other indications for thoracic aortography.

Results

There were 99 patients from the total of 2,205 who suffered a fracture of the sternum. In the group with sternal fracture, there were 2 patients with aortic rupture (2.0%). In the blunt chest injury patients without sternal

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Table 2. Ages of Patients With Sternal Fracture

Age (yr)	No. of Patients With Blunt Chest Injury and Sternal Fracture	No. of Patients With Blunt Chest Injury and No Sternal Fracture
0–9	0	75
10–19	4	307
20–29	25	529
30–39	10	232
40–49	16	241
50–59	16	264
60–69	13	232
70–79	9	138
>80	6	88
Total	99	2,106

fracture, there were 75 patients with TAR (3.6%). These results were not statistically significant ($p = 0.326$).

To our knowledge no patient died in the postinjury period of unrecognized aortic rupture, and none showed enlargement of the aortic shadow on follow-up chest roentgenogram. One patient, however, was seen at another hospital several years after injury with dysphagia and a normal chest film. The workup demonstrated the cause of the dysphagia to be a small pseudoaneurysm of the aorta just distal to the left subclavian artery. The pseudoaneurysm was presumed to have been a consequence of the blunt thoracic trauma originally treated at our institution. The patient was treated successfully with a replacement graft at the other hospital. It is not possible to state with certainty that no other patients in the group "without TAR" did not later show evidence of TAR. This 1 later identified case of TAR without sternal fracture did not alter the statistical analysis.

Comment

Gibson and coworkers [1] were the first investigators to emphasize the association of sternal fractures with other severe injuries in a large series of patients. However, they did not compare patients with sternal fractures with those without them to determine whether the association was significant. Kirsh, Richardson, Naclerio, Zuidema, and associates [2–5], in their trauma texts, reiterated the association of sternal fractures with TAR; each study suggests that sternal fracture may be a predictor of aortic injury and, in and of itself, may be an indication for aortography.

Harley and Mena [7] published a study of 150 patients with blunt chest trauma and noted that 12 of these 150 patients incurred sternal fractures. Of the 12 with sternal fractures, 2 were also noted to have widened mediastinums. These 2 patients suffered aortic injuries. Harley and Mena suggest that sternal fracture in association with widened mediastinum is an indication for aortography; however, note that their conclusion lacks statistical confirmation. No data were given on the remaining 138 patients without sternal fractures, and no statistical comparison was made to determine if the occurrence of thoracic aortic injuries was significantly associated with TAR. Buckman and colleagues [8] reported 60 patients with sternal fractures, none of whom suffered TAR. Wojcik and Morgan [9] recently reported 66 patients with sternal fractures and noted that none of their patients suffered TAR. They pointed out that their findings differed from those of Harley and Mena.

Our data indicate that TAR does not occur more frequently in patients with sternal fractures and blunt chest injuries when compared with patients with blunt chest injuries and no sternal fractures. This interpretation assumes that none of the patients who did not undergo aortography had unrecognized TAR. This assumption is made in light of the further assumption that patients who recover from their injuries fully, and who have normal chest roentgenograms at discharge, did not suffer TAR. (One such case of unrecognized TAR did occur, however.) There exists a degree of speculation in those assumptions, and the reader should interpret our data and conclusions cautiously.

Our investigation is the largest series of patients in whom the frequency of occurrence of TAR has been compared in a concurrent series of patients with blunt chest injury with and without sternal fractures. The lack of a statistical correlation between TAR and sternal fractures should clarify the previously held assumptions that sternal fractures portend aortic rupture, and that sternal fracture is an indication for thoracic angiography in and of itself.

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Pulmonary Artery Versus Left Ventricular Venting: A Radioisotope Study of Left Ventricular Function

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Radionuclide measurements of ejection fraction were used to assess immediate and late postoperative ventricular function after the use of either a pulmonary artery vent (group A) or a left ventricular vent (group B) in 20 patients undergoing aortic valve replacement for pure aortic stenosis. Ten patients were included in each group and anesthetic techniques, patient management, and septal temperatures were similar in all cases. No significant difference was found between the preoperative and immediate or 6-week postoperative ejection fractions, either taken overall or between the two groups ($p > 0.05$; Student's t test). No correlation was found between cross-clamp time, bypass time, or the occurrence of ventricular fibrillation and the immediate postoperative ejection fraction ($p > 0.05$; Student's t test). There was no significant difference in the incidence of ventricular fibrillation after each type of vent had been used ($\chi_0^1 = 3.32$; $p > 0.05$). We did not demonstrate any abnormalities in regional wall motion associated with apical insertion of a left ventricular vent, and conclude that pulmonary artery and left ventricular vents are equally satisfactory in terms of postoperative ventricular performance.

(Ann Thorac Surg 1989;48:699–703)

The question of whether or not to vent the heart during bypass procedures is controversial. In 1976 a survey of surgeons in the United States showed that 64% always used a vent, 17% never used a vent, and 19% sometimes did [1]. The advantages of venting are threefold. First, in open procedures it keeps the field relatively bloodless and improves visibility, whereas in closed procedures it keeps the heart flaccid and improves exposure, particularly when the heart is dislocated as, for example, with operations on the circumflex artery. Second, as the heart is kept empty, rewarming of the myocardium is slowed. Third, venting prevents ventricular distention; this may not necessarily be harmful if it occurs during the period of ischemic arrest [2], but certainly is harmful during reperfusion, where it leads to a reduced ventricular performance [3]. Overdistention during reperfusion reduces subendocardial perfusion, just at a time when the myocardial oxygen demand is high; there is a postischemic oxygen debt to be repayed, and while the heart rewarms there may be a period of ventricular fibrillation (redistributing coronary flow away from the subendocardium [4]). In addition, once contraction becomes normal, if there is overdistention there will be an increased work load associated with the greater ventricular wall stress.

In the past, vents have usually been positioned in the left ventricle, either by direct puncture of the apex of the heart or indirectly by insertion of a catheter through the left atrium or superior pulmonary vein and across the mitral valve. However, left ventricular venting has disadvantages, such as damage to the ventricle or mitral valve, and more recently the use of a pulmonary artery vent has been described [5, 6]. This is claimed to be virtually free of complications [7] and has therefore received support as a better method of venting the heart.

The aim of this study is to use radionuclide scans to measure the ejection fraction in the immediate and late postoperative period as an index of left ventricular function and of myocardial protection afforded by each type of vent. In addition, regional wall motion was assessed visually to determine whether there is local impairment of myocardial contractility at the apex of the left ventricle if a vent is inserted by direct puncture through this site.

Material and Methods

Patients

To eliminate any contribution to myocardial contractility caused by coronary artery disease and ischemia, only patients with pure aortic stenosis and angiographically normal coronary arteries were studied. Those with a previous infarction and those with aortic incompetence were eliminated, as these factors might distort measurement of the ejection fraction.

Twenty consecutive patients meeting the above criteria and undergoing aortic valve replacement were entered into the study after informed consent had been obtained. The patients were randomly divided into two groups: group A, having a pulmonary artery vent; and group B, having a left ventricular vent (inserted through the apex of the heart).

Group A comprised 10 patients (4 male, 6 female; mean age, 66.3 years; range, 50 to 76 years) and group B comprised 10 patients (5 male, 5 female; mean age, 61.7 years; range, 37 to 80 years). All patients underwent routine preoperative investigation, including electrocardiogram; chest roentgenogram; hemoglobin, urea and electrolyte, and serum creatine phosphokinase measurement; echocardiography; and angiography.

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Radionuclide Scanning

Gated blood pool scanning was carried out using in vivo erythrocyte labeling. Patients were injected first with 1.5 mg stannous pyrophosphate and 20 minutes later with 700 MBq technetium 99m pertechnetate. Images were then acquired on an IGE Medical Systems 420 Mobile Gamma Camera, equipped with a slant-hole collimator. Three independent left anterior oblique views were acquired plus one right anterior oblique view. Scanning was performed on the day before the operation and immediately upon return to the intensive care unit after operation. A follow-up scan was performed at 6 weeks. From the left anterior oblique views three measurements of ejection fraction were made and the mean value was taken. All views were used to visually assess regional wall motion.

Valve Replacement

Standard anesthetic techniques were used, employing a narcotic, nitrous oxide, and relaxant sequence; bypass was established using a single right atrial basket and aortic cannula (28FG Whistle Tip, Wessex Medical, England) and a sump catheter, inserted either into the proximal pulmonary artery or into the left ventricular apex (Ross patten vent, G.U. Manufacturing Co, London, England). A COBE CML IF (COBE Laboratories, Gloucester, England) membrane oxygenator was used and a similar perfusion pressure was maintained in all cases. The patient was cooled to 32°C. Once the cross-clamp was applied, the heart was stopped and cooled with 1 L of cold St. Thomas' Hospital cardioplegic solution. Septal temperature was maintained below 25°C using a needle thermometer (type 500 probe; Yellow Springs Instrument Co, OH) and cold topical saline. During bypass the vent output was monitored, and suction was maintained to ensure that the heart remained flaccid. The aortic valve was excised and replaced, using standard techniques, with either a Carpentier-Edwards tissue valve (Baxter Healthcare Ltd, Thetford, England) or a Medtronic prosthesis (Medtronic, Leeds, England).

The heart was rewarmed, air was removed from it, and bypass was discontinued. The pulmonary artery vent site was repaired with a pursestring suture and the left ventricular vent site, by interrupted stitches. Inotropic agents were required in the operating room on six occasions, but were discontinued before arrival in the intensive care unit in all but 1 case. Postoperative hypertension was controlled with vasodilators to maintain the blood pressure within a standard range (specific measurement of systemic vascular resistance was not made). Electrocardiograms were performed on day 1, on day 7, and at 6 weeks. Serum creatine phosphokinase was measured four hours after operation and again at 24 and 48 hours.

Results

In all 20 patients, venting was sufficient to prevent ventricular distention. Patients in group A had a slightly shorter aortic cross-clamp time (mean ± standard deviation, 56 ± 7 minutes) compared with those in group B (mean ± standard deviation, 75 ± 9 minutes). However, total bypass time was longer in group A than in group B (mean ± standard deviation, 75 ± 9 versus 66 ± 20 minutes, respectively).

Table 1. Ejection Fraction Measurements in Patients Undergoing Pulmonary Artery Venting (Group A)

	Ejection Fraction (%)			Bypass Time	Cross-Clamp Time	
Patient	Pre-Op	1-Hour	6-Week	(min)	(min)	Ventricular Fibrillation
LB	43	42	58	65	46	. . .
MC	53	55	48	59	49	. . .
LB	53	53	55	67	50	Yes
WP	54	60	40	85	68	. . .
HS	57	64	59	86	59	Yes
AK	61	53	60	73	57	. . .
HC	62	59	60	82	63	Yes
NJ	63	60	59	77	58	. . .
LT	63	64	71	72	51	Yes
GK	64	. . .	60	84	57	. . .

The hearts of 8 of 10 patients in group B restarted in ventricular fibrillation after the cross-clamp was removed, compared with 4 of 10 in group A. No patient had electrocardiographic evidence of an infarct, and in only 1 patient (in group A) did the serum creatine phosphokinase level rise abnormally.

Septicemia and a paraprosthetic leak developed in one patient in group B 1 month after operation; late follow-up radionuclide scanning was therefore not performed. One further patient in group B sustained a dilated ventricle and increasing myocardial failure after discharge from the hospital and subsequently died 2 months after operation; interpretation of the 6-week ejection fraction measurement is therefore difficult. In 1 patient in group A, the immediate postoperative measurement of ejection fraction was unavoidably delayed for several hours and has therefore been omitted from the results. One patient in group B (MP) had a particularly low ejection fraction at 6 weeks. The reason for this is unclear, as clinically she improved after aortic valve replacement and has remained well since, with no evidence of reduced myocardial performance.

The mean left ventricular ejection fractions for all patients, as well as bypass and cross-clamp times, are shown in Tables 1 and 2.

There was no significant difference in ejection fraction between the preoperative and the immediate postoperative measurements overall, or between the pulmonary artery and left ventricular vent groups ($p > 0.05$, Student's *t* test). Similarly, there was no significant difference in ejection fraction between the preoperative and the 6-week measurements overall, or between the pulmonary artery and left ventricular vent groups ($p > 0.05$, Student's *t* test) (Fig 1). In addition, we found no correlation between cross-clamp time or bypass time and the immediate postoperative ejection fraction ($r = 0.278$ and 0.244, respectively; $p > 0.05$, Student's *t* test).

There was no significant difference in the incidence of ventricular fibrillation after each type of vent had been used ($\chi^1_0 = 3.32$; $p > 0.05$), and analysis of covariance did not demonstrate any significant difference in mean (immediate) ejection fraction measurement between those patients in whom ventricular fibrillation developed and those in whom it did not.

Visual assessment of regional wall motion did not demonstrate any postoperative apical defects in patients having a left ventricular vent (or, indeed, in those with a pulmonary artery vent).

Comment

The use of a left ventricular vent has several disadvantages. If inserted through the right superior pulmonary vein, it can be time-consuming, and whether inserted in this manner or via the left atrium or apex of the ventricle, it requires more retraction of the heart than the insertion of a pulmonary artery vent. Furthermore, it may damage the left atrium, ventricle, or mitral valve, and if inserted through the left ventricular apex, it may disrupt collaterals and may be associated with local impairment of myocardial contractility [8]. Postoperative hemorrhage, aneurysm formation, or air embolism may occur. In contrast, a pulmonary artery vent is virtually free of these complications [7], and provided a sump catheter is used it will eliminate the risk of negative pressure developing and reduce the likelihood of air embolism, particularly in "open" procedures.

Decompression of the left ventricle with a pulmonary artery vent is dependent upon having an incompetent mitral valve. This situation is known to occur during cardioplegic arrest, and it has been shown in both dogs and humans that retrograde flow from the left ventricle to the right side of the heart does occur during ventricular fibrillation [9, 10]. Although the use of a pulmonary artery catheter is known to provide an effective method of venting the heart [5, 6], there has been no direct comparison of postoperative left ventricular function after different types of vent have been used. As the ejection fraction fluctuates less than the cardiac output [11] and is easily measured by noninvasive techniques in the intensive care unit, it was used as an indicator of ventricular performance. In addition, unlike echocardiography, radionuclide measurements of ejection fraction and contractility are not influenced by abnormal septal motion.

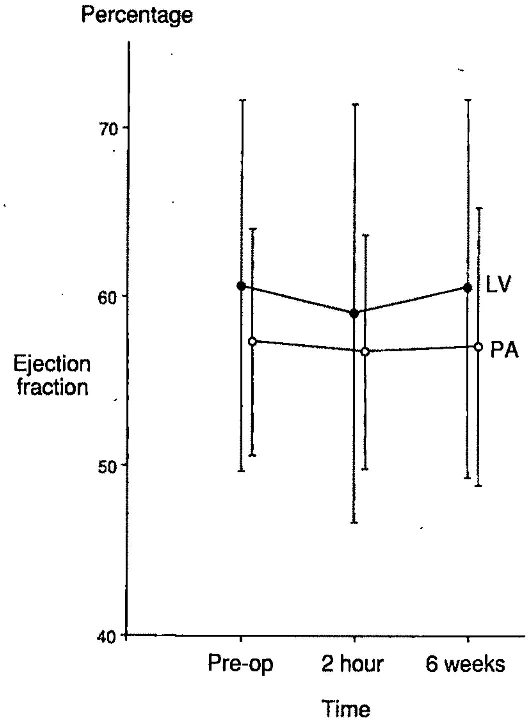

Fig 1. Mean ejection fraction measurements in 20 patients undergoing aortic valve replacement using a pulmonary artery (PA) vent or a left ventricular (LV) vent.

Table 2. Ejection Fraction Measurements in Patients Undergoing Left Ventricular Venting (Group B)

	Ejection Fraction (%)			Bypass Time	Cross-Clamp Time	Ventricular
Patient	Pre-Op	1-Hour	6-Week	(min)	(min)	Fibrillation
KK	43	37	14	45	26	. . .[a]
WS	46	53	59	85	53	Yes
VS	51	55	47	80	63	Yes
JM	54	43	. . .	61	37	. . .[b]
DT	61	66	71	40	25	Yes
PW	66	59	66	80	57	Yes
GW	67	75	61	57	35	Yes
JD	72	60	71	45	32	Yes
RM	72	71	68	67	52	Yes
MP	74	71	41	102	78	Yes

[a] Died at 2 months; [b] Endocarditis and paraprosthetic leak developed in this patient.

There have been several previous reports of ejection fraction measurement in the immediate postoperative period after coronary artery bypass operations. In one, a 17% reduction in mean ejection fraction was observed at one to five hours [12], and in a second study a 24% reduction was observed two hours after operation [13]. In both series the ejection fraction had returned to normal within 24 to 48 hours, and in the later series an actual increase in ejection fraction was observed 1 week postoperatively. Ellis and co-workers [14] showed no change in ejection fraction at 48 hours, and there are at least three series that document ejection fractions being unchanged from preoperative levels at seven to ten days after coronary artery revascularization [15–17]. Taken together, these results suggest that any change in myocardial contractility, as assessed by measurement of ejection fraction, may only be apparent in the first few hours after operation. It is likely that the explanation for this is multifactorial, perhaps related to preoperative drug therapy, the anesthetic technique, the method of myocardial protection, the aortic cross-clamp time, perioperative management, the control of arrhythmias, or the use of inotropic agents.

The series discussed so far concern ejection fraction measurements in patients undergoing coronary revascularization, and it is possible that the transient postoperative decrease observed in two of these series [12, 13] is related to myocardial ischemia, rather than surgical techniques. However, Covitz and associates [11] have also found a significant and transient fall in ejection fraction three to five hours postoperatively in 26 children undergoing open heart operations for a wide variety of congenital abnormalities. Furthermore, they also observed a significant fall in ejection fraction when the aortic cross-clamp time was prolonged. It is interesting, however, that none of the children with low ejection fraction measurements postoperatively had any clinical indication of impaired myocardial function as assessed by blood pressure, central venous pressure, or urine output.

Kennedy and colleagues [18] reported a series of 24 patients undergoing aortic valve replacement in whom the ejection fraction was measured (angiographically) preoperatively and at 19 ± 12 months after operation. Those with aortic stenosis showed no change in ejection fraction, provided this was normal preoperatively; despite correction of the valve abnormality, it would seem unreasonable to expect that this would become better than normal after operation. However, those patients with aortic stenosis and/or aortic regurgitation, who had a low (less than 50%) preoperative ejection fraction, did show significant improvement after valve replacement from 28% ± 8% to 60% ± 16% ($p < 0.01$). Whether this is due to improved ventricular performance after relief of valvular obstruction or due to a reduction in afterload is unclear, but it certainly suggests a potential for improvement in patients with aortic valve disease and a low ejection fraction.

Although only 3 of our patients had a preoperative ejection fraction less than 50%, 2 did markedly improve at 6 weeks, from 43% to 58% and from 46% to 59%. The third patient sustained myocardial failure and died shortly after the 6-week measurement, which accounts for the particularly low value obtained. Excluding these 3 patients, our results are similar to those of Kennedy and colleagues [18] in that we did not demonstrate any change in ejection fraction at 6 weeks in those patients with normal preoperative levels.

In contrast to the findings of Gray and co-workers [12] and Roberts and associates [13] in patients undergoing coronary artery operations and the findings of Covitz and associates [11] in children undergoing repair of congenital heart defects, we did not demonstrate any significant fall in ejection fraction immediately after valve replacement in patients with aortic stenosis. Whether this is a function of differences in technique is unclear. It is impossible to control every variable precisely, but we attempted to achieve this as far as possible by including only patients with pure aortic stenosis and normal coronary arteries, with no evidence of perioperative infarction, and by using similar anesthetic techniques, a standard cardioplegia regimen, temperature monitoring, and similar postoperative care. In this situation we did not demonstrate any significant difference in ejection fraction between those patients having a pulmonary artery vent and those with a left ventricular vent. Furthermore, the type of vent used does not appear to influence the likelihood of experiencing ventricular fibrillation. Patients with poor preoperative left ventricular function represented only 15% of our series. Although they would be expected to derive the most benefit from the use of a vent and may therefore seem the best group to study, changes in left ventricular performance due to changes in operative technique would be more difficult to interpret against a background of a more unpredictable and variable response to implanting a "normal" aortic valve into a severely compromised heart.

In contrast to the results of Covitz and associates [11] and Roberts and colleagues [19], neither the cross-clamp time nor the bypass time influenced the immediate postoperative ejection fraction, and this was independent of the type of vent used. Presumably, the absence of a correlation with cross-clamp time is further evidence of a multifactorial hypothesis to account for any changes in the ejection fraction immediately after operation.

One disadvantage of venting the left ventricle via the apex of the heart is the possibility of subsequent apical dyskinesia. This has been demonstrated in dogs by Sethia and co-workers [8], but in this series the cannulas were still in situ while radionuclide scanning was performed. Nevertheless, Shaw and associates [20], using ventriculograms 9 to 15 months after coronary artery bypass grafting, showed a 56% incidence of apical dyskinesia in 34 patients, compared with a 0% incidence in 14 control patients. However, this group also observed that dyskinesia developed in only 1 patient in 12 with mitral stenosis after valvotomy, performed by inserting a Tubbs dilator through the apex of the heart. It is therefore possible, as these patients are presumed not to have serious coronary artery disease, that it is a combination of an apical vent and ischemic heart disease that is responsible for the focal contraction abnormalities, perhaps a reflection of impaired myocardial healing in the presence of coronary insufficiency. This would be in keeping with our own observations, as none of our patients with pure aortic stenosis (and normal coronary arteries) experienced focal dyskinesia.

In summary, therefore, although postoperative ventricular function is dependent on many variables in patients undergoing aortic valve replacement for pure aortic stenosis, there does not appear to be any difference between using a pulmonary artery vent or a left ventricular vent. However, in view of the greater risk of complications associated with the use of a left ventricular vent, and of the possibility of local impairment of myocardial contractility if the vent is inserted via the apex of the heart in patients with coronary artery disease, we believe that pulmonary artery venting offers substantial advantages and should be the method of choice in most situations.

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Pseudoaneurysm Formation With Superior Vena Caval Syndrome 7 Years After Aortic Composite Graft Placement

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With the advent of the composite graft for combined aortic valve and ascending aortic root operations, postoperative bleeding complications have been reduced. Hemorrhage along the suture sites may occur as a late complication and rarely may originate from the coronary–aortic graft junction. We report a case of a massive pseudoaneurysm, seen as "superior vena caval syndrome," 7 years after operation.

(Ann Thorac Surg 1989;48:704–5)

Introduction of the composite graft for coexistent ascending aortic root and aortic valve disease has improved the associated surgical morbidity and mortality [1–6]. Late complications resulting from bleeding along an anastomosis site have been reported [7], and rarely may involve the coronary anastomosis site [6–8]. We believe that this case, which was seen as superior vena caval syndrome, represents the most dramatic example of this late complication, with massive pseudoaneurysm formation more than 7 years after operation.

A 60-year-old man had been in excellent health until two days before admission to the hospital, when he noted edema and cyanosis of his face and neck. He complained of vague back pain but denied chest pain, shortness of breath, diaphoresis, or lower extremity edema. Past medical history included mixed aortic valve disease, probably rheumatic in origin, and aortic annuloectasia, treated 7 years before admission with a Björk-Shiley prosthesis and composite aortic graft. Because of preexisting coronary disease, saphenous vein bypass grafts were placed to the left anterior descending and obtuse marginal arteries. In addition, the native coronary ostia were reimplanted into the composite graft. Finally, the graft was wrapped with the native aorta, which was sewn closed.

Physical examination showed the patient to be alert and oriented and in no apparent distress. His vital signs included a blood pressure of 110/70 mm Hg in both arms, a regular pulse of 70 beats per minute, and normal respirations and temperature. Examination of the head and neck disclosed jugular venous distention to the angle of the jaw. Cardiovascular examination showed a regular rhythm with normal aortic prosthetic sounds and a 2/6 early systolic ejection murmur, radiating to the carotids. The rest of the examination was unremarkable.

The chest roentgenogram showed mediastinal widening, and the electrocardiogram showed a normal sinus rhythm with inferior Q waves, unchanged from previous tracings. An echocardiogram showed an enlarged ascending aorta with extrinsic encroachment of the right ventricle and right atrium, and a computed tomographic scan confirmed the presence of a large mass adjacent to the ascending aorta which compressed the superior vena cava (Fig 1). Coronary angiography and arch studies showed no aneurysmal dilatation of the composite graft and a normally functioning prosthetic valve and patent coronary artery grafts.

At operation, a clot-filled pseudoaneurysm was discovered with persistent bleeding from the left coronary artery to the composite graft anastomotic site. Attempts to repair the composite graft were unsuccessful, and the patient died during operation.

Comment

Surgical management of aortic root aneurysms and associated aortic valve disease has traditionally been a difficult problem. With the advent of the composite graft in 1968 [1], operative mortality and postoperative complications have been dramatically reduced [2–6]. This procedure and its modifications replace the ascending aorta to the level of the annulus with a composite unit of valved tubular graft, onto which the coronary ostia are reanastomosed. The advantages include reduction in subsequent aortic aneurysms and a lower incidence of postoperative bleeding and paravalvular leaks.

A late complication of the operation is hemorrhage along a suture line, with the potential for pseudoaneurysm formation. Such pseudoaneurysms tend to originate from the proximal or distal aortic suture lines [7], but infrequently arise from the site of coronary anastomoses. We believe that our case is unique for the size of the pseudoaneurysm, originating from the coronary ostial site so late after operation, and appearing only as superior vena caval syndrome. It also emphasizes the utility of computed tomographic scanning in localizing the extent of the complication.

Bleeding from the coronary anastomosis after composite graft operations has been reported, and techniques to reduce the complication have been proposed. Among 127

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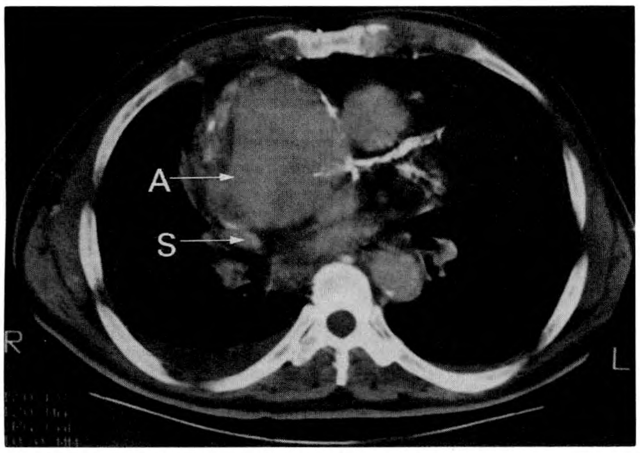

Fig 1. Computed tomographic scan disclosed the large pseudoaneurysm (A) originating from the left coronary artery to graft junction, with compression of the superior vena cava (S).

patients undergoing composite graft replacement of the ascending aorta and aortic valve, Kouchoukos and colleagues [5] reported 4 patients in whom small pseudoaneurysms developed from a coronary ostial site early after operation, 1 of whom underwent reoperation for chest pain. McCready and Pluth [6] also reported 2 patients with small pseudoaneurysms from coronary sites, both of which were incidentally found on angiography. Mayer and associates [8] identified 2 patients with pseudoaneurysms originating from the site of coronary ostial implantation at least 1 year postoperatively; they identified the inferior position as the site of suture line dehiscence and advocate buttressing the coronary ostial suture line at that site with a Teflon felt buttress. In addition, to alleviate tension along the anastomoses and reduce bleeding complications, interposition of saphenous vein grafts or Dacron grafts [3] has shown good results.

Bleeding from the coronary ostial anastomosis can occur long after an aortic composite graft operation. We believe that our case is a dramatic example of this, with a massive pseudoaneurysm seen as superior vena caval syndrome more than 7 years postoperatively. Frequent follow-up in such patients is vital, particularly when the inclusive (wrap) technique has been used.

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Use of Pericardium to Control Bleeding After Ascending Aortic Graft Replacement

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We present the case of a patient who required replacement of an ascending aortic composite graft for endocarditis complicated by uncontrollable bleeding from the proximal anastomotic site. A new method of creating a right atrial-to-periprosthetic space fistula with autologous pericardium was used with a favorable result.

(Ann Thorac Surg 1989;48:706–8)

Treatment of aortic insufficiency associated with composite aortic graft endocarditis has traditionally been a difficult problem [1]. Surgical intervention is frequently complicated by persistent bleeding from the proximal composite graft anastomosis [2–4], especially in the presence of abnormal coagulation associated with renal failure [5]. The recently introduced method of wrapping the composite graft with native aorta and producing a fistula between the right atrium and periprosthetic space has been advocated as a method of dealing with persistent bleeding [2, 3], but this technique cannot be used when native aorta is unavailable due to a previous operation or scarring. We present a method of using the pericardium to form a pouch around the proximal composite graft anastomosis, which allows development of a right atrial-to-periprosthetic space fistula when native aorta is unavailable.

A 70-year-old man with Marfan's syndrome who had undergone composite ascending aortic graft placement 8 years earlier for ascending aortic aneurysm with severe aortic insufficiency was seen with acute bacterial infection of his composite graft and aortic insufficiency. On transfer from an outside hospital, he was in florid congestive heart failure; his blood pressure was 135/30 mm Hg on inotropic support. His pulmonary capillary wedge pressure was 30 mm Hg with pulmonary arterial pressures of 50/17 mm Hg. He was in acute renal failure, with a blood urea nitrogen level of 102 mg/dL and creatinine level of 6.1 mg/dL. Preoperative echocardiography revealed serious aortic insufficiency with an enlarged left ventricular cavity and mild mitral regurgitation. A lucent halo was suggestive of an aortic root abscess. His temperature was 104°C, and his white blood cell count was 35,000/μL with a leftward shift. His urinalysis revealed proteinuria and gross hematuria. He was believed to have severe immune complex–induced renal failure and severe aortic insufficiency, both secondary to an infected aortic composite graft.

The patient underwent preoperative hemodialysis and operation for replacement of his infected graft eight hours after his admission on July 7, 1988. Because of his tenuous hemodynamic state, before endotracheal intubation, normothermic cardiopulmonary bypass was initiated under local anesthesia through his femoral vessels at a rate of 2 L/min. When his median sternotomy incision was reopened, multiple dense adhesions were encountered. After aortic cross-clamping, we performed an arteriotomy into the composite graft. A large pocket of pus in the periprosthetic space (which subsequently grew *Staphylococcus epidermitus*) surrounded the entire aortic root and both coronary ostial anastomoses.

Numerous vegetations were found on the aortic valve, with tears in all three leaflets of the previously placed Hancock bioprosthesis. The entire graft and valve were removed, with care taken to preserve the fibrous margin of the left and right coronary ostia. Removal of the sewing ring of the prosthesis revealed an entire circumferential annular abscess resulting in dehiscence of the mitral annulus from the aortic root. This was liberally irrigated with a saline solution and debrided of necrotic material. Because of the rigid nature of the superiorly displaced aortic root structures, the Cabrol technique [2, 3] was used: an interposition graft of 8-mm Dacron to communicate the two coronary ostia was placed with a continuous 4-0 polypropylene suture (Prolene; Ethicon, Somerville, NJ) (Fig 1). Circumferential sutures of 3-0 polypropylene were then placed in the aortic annulus to obliterate the abscess cavity and to reapproximate the mitral annulus to the aortic annulus. A 31-mm St. Jude composite graft was then sutured in place. A side-to-side 1.5-cm anastomosis between the aortic root and 8-mm Dacron graft was then made with continuous 4-0 polypropylene. The distal aortic anastomosis was made with a continuous 3-0 Prolene suture reinforced with Teflon felt.

As the cross-clamp was released after air was removed from the graft, massive hemorrhage from the proximal anastomosis was noted. The aortic annulus appeared to be starting to disintegrate, resulting in tearing of the friable necrotic tissues used to sew the composite graft. Multiple sutures were placed, with generous reinforcement with Teflon felt pledgets. Nevertheless, persistent, inaccessible bleeding resulted in further tearing of the tissues. As a final effort, a flap of right lateral pericardium

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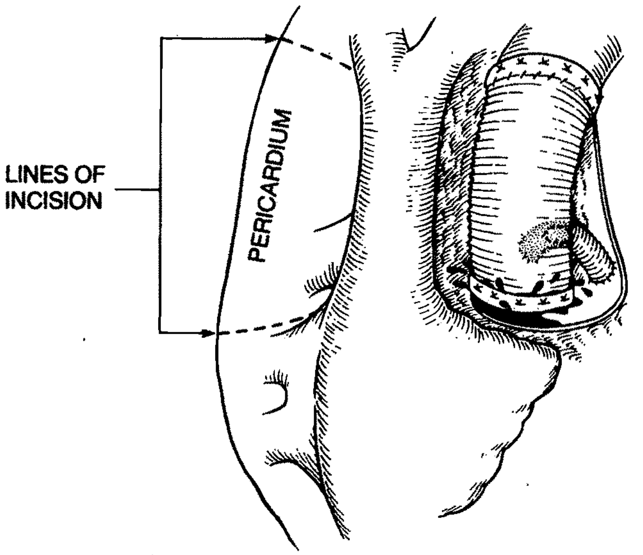

Fig 1. Reimplanted composite graft conduit inserted using Cabrol method for infected aortic root graft with necrotic annulus and massive bleeding from annular suture line. Incisions in pericardium are shown by arrows.

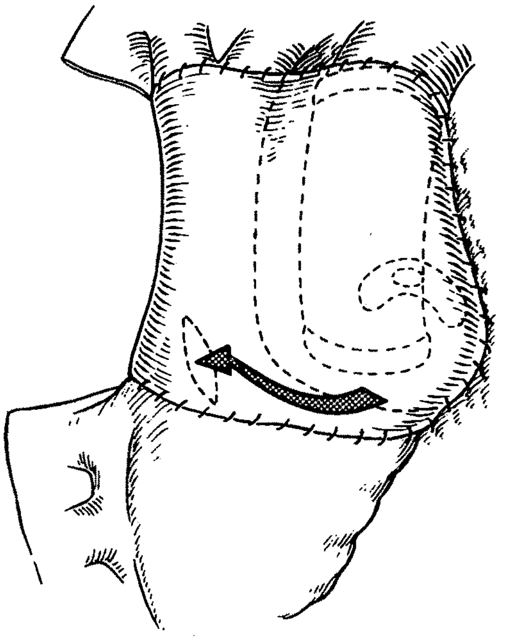

Fig 3. Completed pericardial flap coverage of the entire aortic root. Arrow *indicates fistula between periprosthetic space and right atrium.*

was mobilized and sutured along the superior vena cava, the superior margin of the right atrium, the anterior aorta, and down to the left of the aortic root (Fig 2). Once the patch was nearly completed, a 1.0-cm fistula was made in the roof of the right atrium (Fig 3). The proximal suture line was completed, and bleeding was satisfactorily controlled. The patient did surprisingly well postoperatively, but required renal dialysis treatments. He was discharged on postoperative day 45. An echocardiogram obtained before discharge revealed a small pericardial effusion and a patent right atrial fistula. The patient died 4 months postoperatively of a ruptured right iliac artery aneurysm. Just before his death, his activity level was New York Heart Association class I.

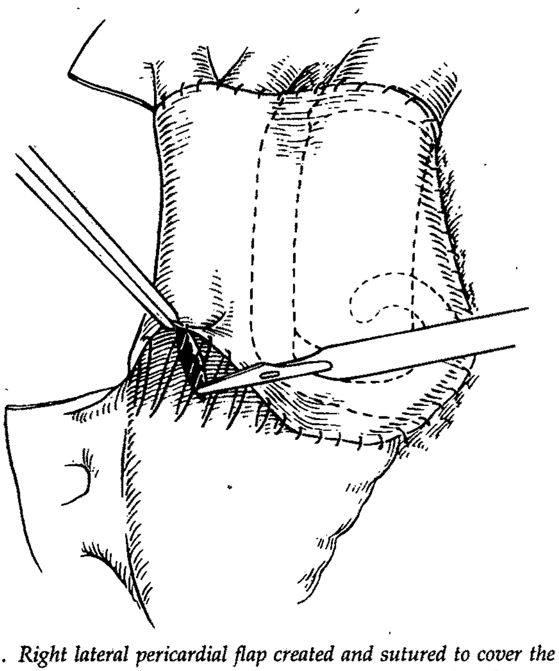

Fig 2. Right lateral pericardial flap created and sutured to cover the entire aortic root. Right atrial incision was made just before the suture line was completed.

Comment

Had it been available, our preferred method of treatment would have been an aortic homograft with exclusion of the infected root abscess from the bloodstream [6]. However, we were forced to use a prosthetic composite graft instead. Bleeding complications with ascending aortic root replacement have prompted development of techniques by which the graft is wrapped with the remaining native aorta to control bleeding [7]. Cabrol and colleagues [2] extended this idea by creating a fistula between the periprosthetic space and the right atrium to prevent development of a hematoma under tension with eventual tearing of the aortic wall. Long-term follow-up with this technique has been excellent [3]. The Cabrol technique has been criticized, however, because of the potential dangers involved in mobilizing a proximal aortic root that is rigid due to a previous operation or scarring [8]. Our technique avoids these hazards and can be used when the native aortic root has been resected. Autologous pericardium is easy to use, free of cost, readily available, and pliable enough to occlude the interstices, thus increasing hemostasis, yet it is strong enough to buttress sutures [9]. Furthermore, if autologous pericardium is unavailable, glutaraldehyde-preserved bovine pericardium may prove a suitable alternative when one forms a periprosthetic pouch-to-right atria fistula with this technique.

Despite our patient's death 4 months after operation, we were able to wean him from bypass using our technique and to discharge him from the hospital. Hemodynamically and functionally, he did quite well until he died of another cause. The long-term fate of his internal right atrial fistula is not known but is of concern. Cabrol and colleagues [3] commented that periprosthetic-to-right atrial fistulas usually close soon after operation; however, they can be surgically closed relatively easily if they become hemodynamically significant. In our patient, the internal fistula would have been more difficult to close because closing it would have necessitated opening the autologous pericardial pouch to suture the right atrial fistula closed directly.

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Modified Cabrol Shunt for Control of Hemorrhage in Repair of Type A Dissection of the Aorta

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A technique of modifying the Cabrol shunt using preserved bovine pericardium and a patch of autologous pericardium to deal with postoperative hemorrhage from an ascending aortic operation is described. A fistula to the right atrium was created for autotransfusion. This simple technique is very useful for dealing with the catastrophic complication of such postoperative hemorrhage.

(Ann Thorac Surg 1989;48:709–11)

During repair of an acute type A dissection of the aorta involving the arch vessels, severe hemorrhage occurred at the anastomotic sites, which was difficult to control by conventional measures. We describe a successfully applied modification of the Cabrol shunt utilizing autogenous and bovine pericardium.

Case Presentation and Technique

A 56-year-old man with a one-hour history of sharp retrosternal chest pain radiating to the upper midback collapsed and was brought to the emergency department. Physical examination showed him to be in moderate distress but alert and oriented. His blood pressure was 170/80 mm Hg in the left arm and 60 mm Hg systolic in the right arm. His heart rate was 100 beats/min. Heart sounds were normal, and there were no murmurs. The extremities were well perfused. A chest roentgenogram showed a wide aortic arch. A left radial arterial line was inserted, and intravenous nitroprusside and Inderal (Ayerst Laboratories, New York, NY) were administered to control hypertension.

Doppler echocardiography showed a flap in the ascending aorta with a trace of aortic insufficiency. The patient remained hemodynamically stable and was further investigated with computed tomographic scan and angiography as part of a clinical investigative protocol. Noncontrast dynamic computed tomographic scan showed a 7-cm diameter aortic root. A well-defined intimal flap was seen in the lumen of the ascending aorta extending into the arch as far as the left subclavian artery. The descending thoracic aorta was normal. A pericardial effusion was noted. Aortography (Fig 1) confirmed the presence of type A dissection. The intimal tear was not seen. The innominate artery was cut off 2.5 cm from its origin. A flap was seen in the left common carotid artery. The left subclavian artery was not visualized. There was no evidence of aortic insufficiency, and the coronary arteries were not injected.

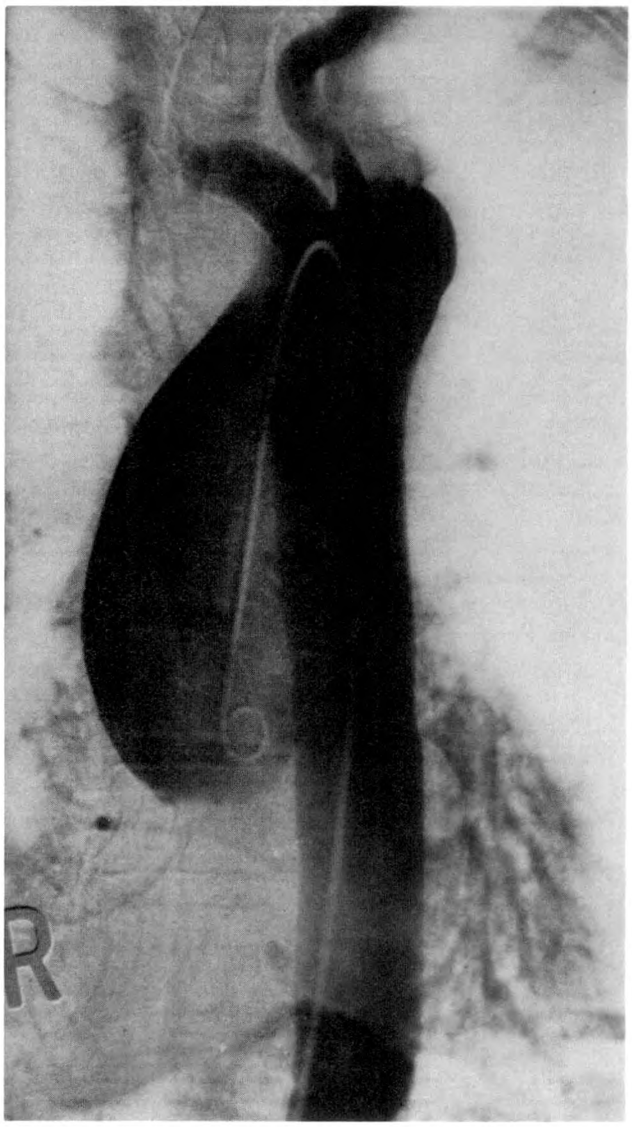

Fig 1. The intimal flap of the ascending aortic dissection is shown in the aortogram adjacent to the tip of the angiographic catheter.

At operation, the patient was heparinized, and the left common femoral artery was cannulated. The femoral

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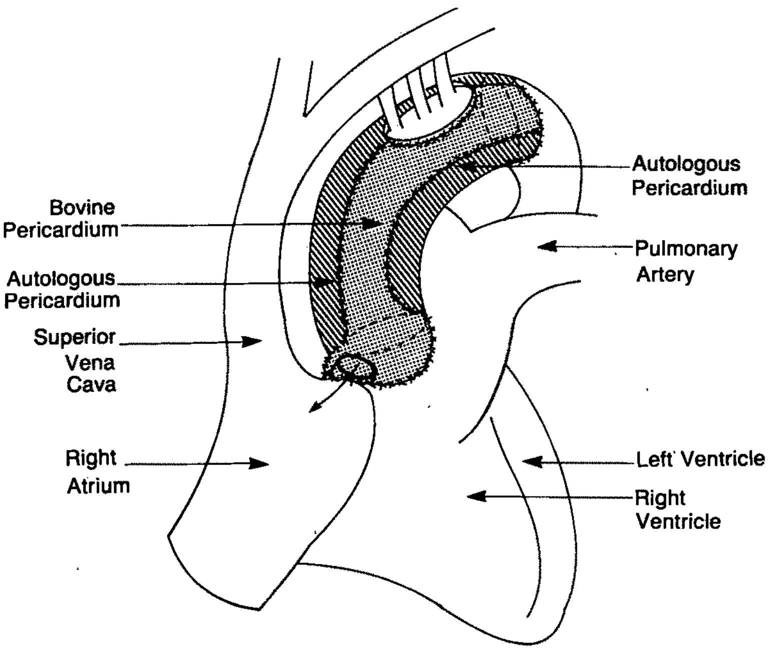
Fig 2. The modified Cabrol shunt. Two pieces of pericardium (autologous and bovine) are used to create the aortoatrial fistula.



artery was slightly atherosclerotic, but the aortic dissection did not extend into the femoral artery. A median sternotomy was performed, and a tense pericardium contained 500 mL of fresh blood. The dissection extended proximally into the base of the aorta with blood in the epicardial fat. The right atrium was cannulated, and cardiopulmonary bypass was instituted. Solu-Medrol (Upjohn Co, Kalamazoo, MI) was administered intravenously. Under hypothermic total circulatory arrest (rectal temperature, 14°C), an anterior longitudinal incision was made in the ascending aorta and cannulas were placed in the coronary ostia for infusion of continuous cold blood cardioplegia (40 mL/min).

A circumferential tear was present in the midportion of the ascending aorta. The dissection extended proximally to within 1 cm of the coronary ostia and distally 1 to 2 cm beyond the left subclavian arteries. The false lumen extended into both the innominate and subclavian arteries. The aortic valve was normal. The longitudinal aortotomy was carried from 1 cm above the aortic annulus to 2 cm beyond the left subclavian artery. The proximal aorta was transected circumferentially. All diseased ascending aorta and the concave portion of the arch were excised. An island of the convex arch containing the head vessels was preserved. A preclotted, baked, 28-mm woven Dacron graft was trimmed with a complementary tongue, and the distal aortic anastomosis was performed incorporating the arch vessels in a single suture line of running 3-0 Prolene (Ethicon). Air was removed from the distal aorta, graft, and arch vessels, and the graft was clamped proximal to the innominate artery. Cardiopulmonary bypass was resumed after 33 minutes of circulatory arrest. While the patient was rewarmed, we performed the proximal anastomosis at the aortic annulus, preserving the aortic valve. The graft was sutured to an appropriately tailored aorta incorporating both coronary ostia. Air was removed from the heart, and the cross-clamp was released 25 minutes after the graft was clamped. The heart spontaneously resumed a normal sinus rhythm.

Profuse bleeding occurred from inaccessible areas at the proximal and distal anastomoses posteriorly. Because bleeding persisted despite use of extra pledgeted sutures, weaning from bypass, and administration of protamine sulfate, the patient was reheparinized and bypass was reinstituted. A modified Cabrol shunt was fashioned as follows.

A patch of autogenous pericardium was sutured to the proximal and distal anastomoses, posterior to the graft. With a running 4-0 Prolene suture, the edges of a glutaraldehyde-preserved bovine pericardial patch were sutured to (1) the distal anastomosis, (2) the edge of the patch of autogenous pericardium to the bovine pericardium, and (3) the conus and the right atrium. The small remaining edge of bovine pericardium was sutured over the atriotomy site (Cabrol shunt) after bypass had been discontinued and the right atrium had been decannulated (Fig 2). Hemostasis was secured immediately after administration of protamine. The aorta was decannulated, and the operation was completed in the usual fashion.

The patient was extubated nine hours later. Doppler echocardiography on the second postoperative day showed no flow through the Cabrol shunt. The patient was discharged on the ninth postoperative day. He was well at 3-month follow-up.

Comment

Follow-up studies of patients undergoing the Bentall-DeBono procedure [1] demonstrate a high incidence of pseudoaneurysm formation at one or more of the suture lines [2, 3], which is believed to be related to the wrapping of the aneurysm shell around the graft to achieve hemostasis. Due to continued bleeding into the space between the graft and overlying aorta, excessive tension may develop on the suture lines, especially at the coronary ostium–graft anastomoses. Cabrol and colleagues [4] reported construction of a fistula between the aneurysm wrap and the right atrial appendage to control bleeding (autotransfusion) and to prevent false aneurysm formation.

Kouchoukos and associates [3, 5] have abandoned the inclusion technique in favor of the open technique of aneurysm repair and have observed no pseudoaneurysm formation at the suture line. This has been confirmed by other investigators [6, 7]. The technique is appealing because it allows resection of all diseased aorta, precise suturing, and access to bleeding areas at the end of bypass. However, if troublesome bleeding occurs due to poor tissue quality (as in dissection of the aorta), no wrap is possible because the aneurysm shell or aortic wall has been removed. Kouchoukos [5] disagreed with Marvasti and co-workers [2] as to the usefulness of the aneurysm wrap for hemostasis in ascending aortic repairs. Because most surgeons have now adopted the open technique and remove the diseased aorta, there is no option to wrap the graft with the aneurysm shell or aortic wall.

Longer follow-up and more clinical experience is needed to delineate late complications. The shunt appears to subside a few days postoperatively; therefore, late complications should be similar to those of other reports of the open technique [5–7].

Troublesome bleeding occurred after an open repair of acute type A dissection involving the arch vessels despite precise suturing of the graft. We were able to save the patient by wrapping the graft with autogenous and bovine pericardium, incorporating the suture lines, and creating a modified Cabrol shunt. This technique allows an option for dealing with severe bleeding when the open technique is used for operations on the ascending aorta and arch vessels.

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Ancrod Anticoagulation for Cardiopulmonary Bypass in Heparin-Induced Thrombocytopenia and Thrombosis

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Heparin-induced thrombocytopenia and thrombosis was diagnosed in a 50-year-old man undergoing a repeat heart operation after heparinization led to microemboli and an eventual left transmetatarsal amputation. A third heart operation was aborted when anticoagulation with low molecular weight heparin produced intraoperative thrombi. The patient was referred to Toronto where ancrod (Arvin) was used to lower plasma fibrinogen level, allowing successful repair of a ventricular septal defect using cardiopulmonary bypass support. The patient made an uneventful recovery.

(Ann Thorac Surg 1989;48:712–3)

Thrombocytopenia occurs in up to 15% of patients who receive heparin. Usually this thrombocytopenia is not associated with serious morbidity and resolves after administration of heparin is discontinued. In a small subset of these patients, thrombosis and thromboembolism develop which, even if the heparin is withdrawn as soon as the syndrome is recognized, may progress to strokes, myocardial infarctions, ischemic or infarcted bowel, and massive tissue necrosis of limbs. The estimated morbidity and mortality are 23% and 12%, respectively [1].

Antagonists to vitamin K (eg, warfarin sodium) are safe alternatives, but procedures requiring cardiopulmonary bypass with heparin may be denied to these patients. Because of our early success with ancrod (Arvin, Knoll A.G. Pharmaceuticals, Ludwigshafen, FRG; distributed by Knoll Pharmaceuticals, Markham, Ont, Canada), a defibrinogenating agent derived from Malayan pit viper venom, as an alternative to heparin for cardiopulmonary bypass [2], the patient we describe was referred to us.

A 50-year-old man with a previous history of double aortocoronary bypass grafting in 1980 was admitted to a hospital in North Carolina with an acute myocardial infarction complicated by a ventricular septal defect (VSD) and circulatory failure. He was stabilized using dopamine, nitroprusside, and an intraaortic balloon pump. Heart catheterization demonstrated an occluded right coronary graft, a patent left anterior descending graft, and a large VSD.

On April 30, 1987, the VSD was closed and single aortocoronary bypass grafting to the obtuse marginal artery was performed. A severe thrombocytopenia developed intraoperatively, complicated by microemboli that affected the lower extremities predominantly and necessitated a transmetatarsal amputation of the left forefoot. The diagnosis of heparin-induced thrombocytopenia was made.

On July 20, 1987, the patient was readmitted with a deep vein thrombosis of the left lower extremity. A test dose of heparin was administered, and the platelet count decreased from 3.66 to 1.46 × 10^9/L. The deep vein thrombosis was treated with Coumadin (crystallin warfarin sodium) alone.

The patient was readmitted on October 31, 1987, with increasing angina and heart failure. Repeat cardiac catheterization revealed moderate ventricular dysfunction, a VSD with a 2.3:1 shunt, patent left anterior descending and obtuse marginal grafts, and the previous occluded right coronary graft. Hematocrit was 0.25 because of an intravascular hemolysis attributed to the persistent VSD. After confirmation of heparin-induced thrombocytopenia and thrombosis in vitro, repair of the VSD was attempted with an experimental low molecular weight heparin (ORG-10172). Intraoperatively, thrombi were noted in the extracorporeal circulation despite an adequate activated clotting time; the surgical procedure was promptly aborted. At this stage, the patient was referred to us for cardiac operation with ancrod as the anticoagulant.

At the Toronto General Hospital, two-dimensional echocardiogram confirmed a large posterior VSD, trivial mitral regurgitation, and an akinetic septum and inferior wall. The initial fibrinogen level was 3.4 g/L, platelet count was 2.40 × 10^9/L, and hematocrit was 0.40. On March 3, 1988, 208 U of ancrod were infused over six hours to achieve a plasma fibrinogen level of 0.8 g/L. Eleven units of ancrod were infused over the remaining ten hours before operation, and the patient entered the operating room with a fibrinogen concentration of 0.45 g/L.

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A tedious fourth resternotomy was performed. Surgical technique included bicaval cannulation, aortic arch cannulation, and cooling to 30°C on heart-lung bypass. Remnants of the previous VSD repair were removed, and the defect was patched with thick autogenous pericardium. The heart was defibrillated after a total fibrillation time of 53 minutes. Successful weaning from cardiopulmonary bypass was achieved with an intraaortic balloon pump and low-dose inotropic support. Blood loss in the operating room was estimated at 4,000 mL. Ten units of packed red blood cells was administered intraoperatively. An autotransfusion system was connected to the mediastinal drains at the end of the procedure, as is routine in our hospital.

The immediate postoperative fibrinogen concentration was 0.7 g/L, and the platelet count was 1.10×10^9/L with a hematocrit of 0.34. Twenty-four hours after the operation, the fibrinogen concentration was 1.47 g/L (normal, 1.5 to 2.5 g/L). On the morning of the second postoperative day it was 2.05 g/L. Chest tube drainage over the first 24 hours was 1,490 mL. Six units of packed red blood cells, 6 U of platelet concentrate, and 4 U of plasma were administered in the intensive care unit.

The patient had an uncomplicated recovery in the hospital. A postoperative two-dimensional echocardiogram indicated no residual VSD. Monitoring with iodine 125-labeled fibrinogen and impedance plethysmography showed no deep vein thrombosis throughout the patient's perioperative course. He was fit for discharge after 10 days of hospitalization.

Comment

The need for anticoagulation during cardiopulmonary bypass poses a special problem for patients known to be sensitive to heparin. Other tactics to prevent heparin-induced thrombocytopenia and thrombosis have been directed toward modifying platelet responsiveness. Two approaches have been successful. Treatment with aspirin and dipyridamole before operation [3] or an infusion of a prostacycline analogue (Iloprost [ZK 36374]), administered before and during operation [4], have allowed the use of heparin during cardiopulmonary bypass without complications.

Ancrod acts enzymatically to cleave fibrinogen to split off the A-fibrinopeptides, but not fibrinopeptide B [5], to produce a friable, unstable non–cross-linked particulate fibrin. This imperfectly polymerized fibrin is rapidly removed from the circulation by fibrinolysis, with the formation of fibrin split products, and by reticuloendothelial phagocytosis. Ancrod is believed to stimulate fibrinolytic activity in established thrombi by dose-dependent release of tissue plasminogen activator from the vascular endothelium [6]. It has been used to treat patients with deep vein thrombosis and pulmonary embolism and as postoperative prophylaxis for thromboembolic events [7]. Our preliminary results using ancrod for cardiopulmonary bypass show low fibrinogen levels with no effect on the level or activity of any other clotting factors or any adverse platelet effects.

Characteristic of substances that depend on phagocytosis by the reticuloendothelium system for their elimination, the half-life curves for ancrod are multiexponential. The initial rapid elimination has a half-life of three to five hours, with 90% of the ancrod cleared by four days.

Use of ancrod avoids the risk of allergic and hemodynamic reactions to the protamine [8] required to neutralize heparin; however, ancrod itself is a foreign protein that could be associated with an allergic reaction, although we have not experienced this. Although the need for it is rare, ancrod antidote (manufactured by Knoll from goat serum and distributed by Connaught Laboratories, Toronto, Ont, Canada) is available and is capable of restoring plasma fibrinogen to normal levels within an hour. The antidote may be associated with a foreign protein-type allergic reaction, and intravenous administration of fibrinogen is the preferred method to reverse the effects of ancrod.

This case report describes the successful use of ancrod as an alternative anticoagulant to heparin for cardiopulmonary bypass in a man with life-threatening heparin-induced thrombocytopenia and thrombosis.

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Severe Stenotic Scar Contracture of the Microvel Hemashield Right-Sided Extracardiac Conduit

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We report 2 patients who developed severe scar contracture of a Microvel Hemashield valveless right-sided extracardiac conduit implanted 14 months earlier. In both cases, the diameter of the conduit was reduced along its entire length, with the prosthetic material plicated inward by retraction and organization of connective fibrous tissue on either side of the conduit.

(Ann Thorac Surg 1989;48:714–6)

Collagen-coated vascular prostheses have been advocated for many years for their nonporous surface, which minimizes blood loss at the time of operation, particularly in heparinized patients [1–3]. This, together with the facts that such prostheses are readily available, soft, pliable, and easy to handle, has extended their use, particularly for repair of complex congenital cardiac malformations. We report 2 cases of severe stenotic scar contraction of a Microvel Hemashield prosthesis used as a right-sided extracardiac conduit.

Case Reports

Patient 1

In August 1987, a 7-year-old girl was admitted for elective total repair of double-outlet right ventricle, noncommitted ventricular septal defect (VSD), and pulmonary atresia. At operation, an intraventricular tunnel was created with a Gore-Tex patch from the left ventricle through the VSD into the aorta, and a Microvel Hemashield No. 18 extracardiac conduit was used to connect the right ventricle to the main pulmonary artery. After the operation, the child remained well until November 1987, when she started to complain of dyspnea and reduced exercise tolerance. Echocardiographic examination and subsequent catheterization confirmed the presence of a residual VSD with a substantial left-to-right shunt. No clinically significant gradient was found between the right ventricle and the pulmonary artery.

Angiography showed no narrowing of the lumen of the extracardiac conduit (Fig 1A). At reoperation, the right ventricular anastomosis of the extracardiac conduit was opened to expose the residual VSD. The Gore-Tex tunnel used at the first operation was detached along the ventriculoinfundibular fold from the level of the tricuspid valve to the aorta and also along the lateral edge of the interventricular tunnel. These two dehiscent edges were refixed using interrupted Prolene sutures. Examination of the conduit showed the lumen to be uniformly covered by a thin fibrous layer with no evidence of narrowing or scar contraction. The proximal end of the conduit was therefore reanastomosed to the ventriculotomy. The postoperative period was uncomplicated, and the child remained asymptomatic.

In August 1988 during a routine follow-up visit a new loud left parasternal systolic murmur was discovered. Doppler echocardiography showed severe obstruction of the extracardiac conduit, with a peak velocity systolic flow across the conduit of almost 6 m/s and an estimated gradient of 130 mm Hg. At catheterization under general anesthesia, a 60-mm Hg gradient was found. Angiography showed the diameter of the conduit to be severely reduced along its entire length (Fig 1B). In view of these findings, the child underwent elective reoperation at the beginning of September. At operation, a uniformly stenotic extracardiac conduit was removed and replaced with a pulmonary homograft. The operation was uneventful, and the child was discharged from the hospital on the eleventh postoperative day.

Macroscopic examination of the conduit showed uniform concentric contraction with reduction of the inner diameter from the original 18 mm to 8 mm. Histology of the specimen showed the prosthetic material to be embedded in loosely arranged connective tissue with extensive foreign body giant cell reaction and plicated inward by retraction and organization of connective fibrous tissue on either side of the conduit.

Patient 2

In March 1987, a 12-year-old girl was admitted for elective operation of congenitally corrected transposition of the great arteries, atrial and ventricular septal defects, pulmonary atresia, and closure of a right Blalock shunt. At operation, after closure of the Blalock shunt the VSD was closed with a Gore-Tex patch through the aorta, and the atrial septal defect was closed with autologous pericardium. A Microvel Hemashield No. 20 extracardiac conduit was then used to connect the left ventricle to the main pulmonary artery. After the uneventful operation, the child remained well until March 1988, when she started to complain of tiredness and dyspnea. Echocardiographic examination and subsequent catheterization showed a

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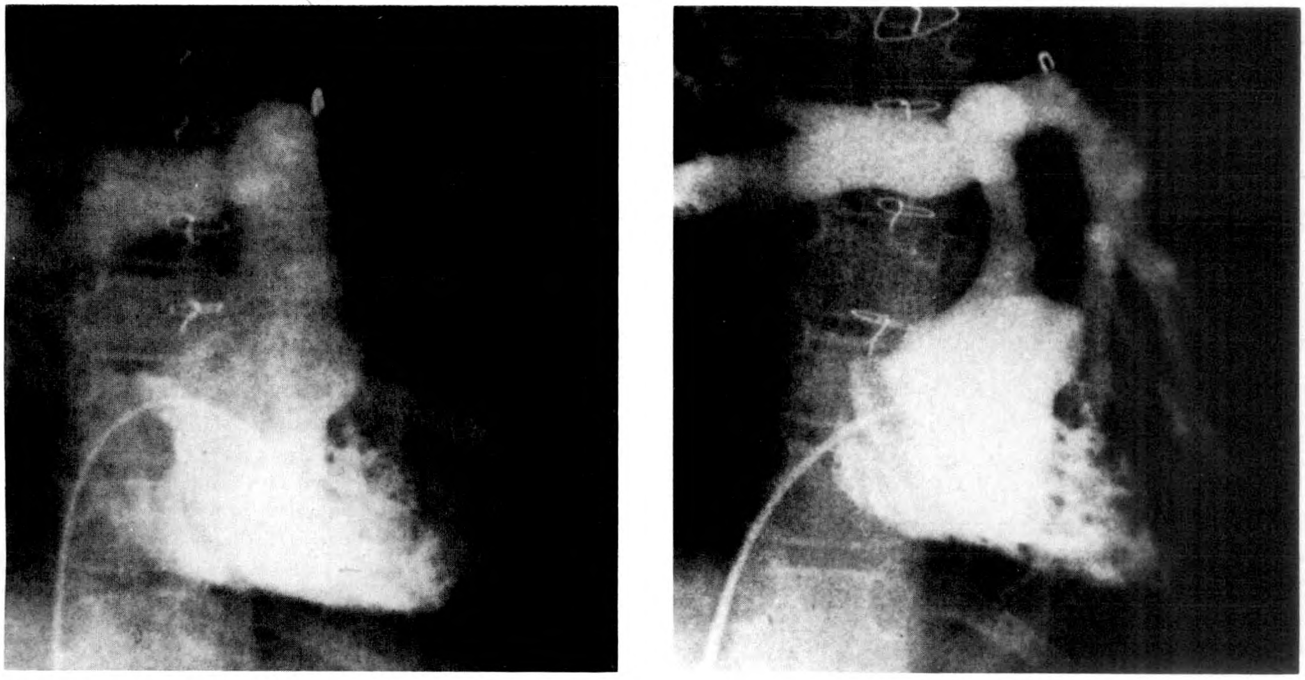

Fig 1. (Patient 1.) Anteroposterior right ventricular angiograms showing (A) normal appearance of the right-sided extracardiac conduit 3 months after implantation and (B) uniformly narrowed right-sided extracardiac conduit 14 months after implantation.

residual VSD together with severe stenosis of the extracardiac conduit. In view of these findings, the child underwent elective reoperation at the beginning of June. At operation, a uniformly contracted stenotic extracardiac conduit was removed and replaced with a pulmonary homograft. The residual VSD was exposed and closed through the aortic valve. The operation was uncomplicated, and the child was discharged from the hospital on the thirteenth postoperative day. Macroscopic examination of the conduit showed inward contraction and substantial uniform narrowing of its inner diameter from the original 20 mm to 9 mm. Histological examination of the specimen showed a foreign body giant cell reaction with extensive fibrous tissue proliferation on either side of the prosthetic material (Fig 2), similar to the findings in patient 1.

Comment

Synthetic vascular prostheses have long been used for correction of complex congenital cardiac malformations [4, 5]. Because these surgical procedures require extracorporeal circulation and systemic heparinization, any conduit used must be hemostatic to avoid postoperative bleeding, which can be difficult to control.

Initially, low-porosity woven Dacron prostheses fulfilled this requirement. However, the nonporous construction of the Dacron contributes to the peculiar, progressive, proliferative luminal fibrous peel responsible for the severe obstruction that occurs in many such conduits [4–6]. Introduction of the Microvel Hemashield vascular prosthesis appeared to be the answer to all of these problems. Made of knitted Dacron double-velour impregnated with absorbable, cross-linked collagen, the Microvel Hemashield has a nonporous surface which makes it impermeable to blood at the time of implantation [1, 7, 8]. After digestion of the collagen, the porous knitted Dacron exposed should prevent any exuberant luminal peel formation. Indeed, in both of our patients, the severe obstruction of the conduit was not due to luminal peel formation, but to scar retraction and inward plication of the Dacron between the connective fibrous tissue developed on either side of the conduit. This was not observed

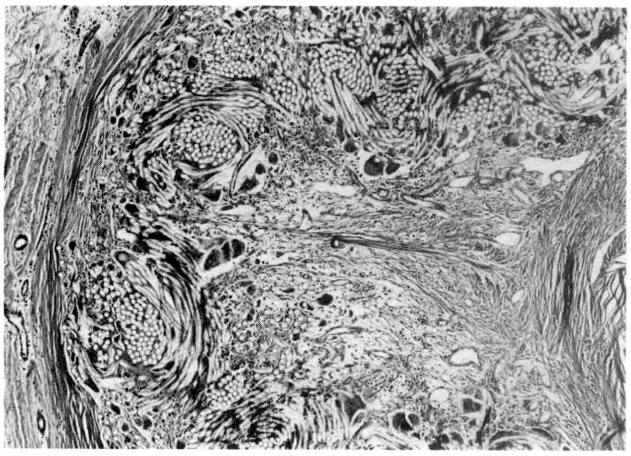

Fig 2. (Patient 2.) Histology of the explanted extracardiac conduit showing the prosthetic material embedded with loosely arranged connective tissue with a foreign body giant cell reaction. The prosthesis is plicated inward by hypocellular connective tissue on either side of the conduit. (Hematoxylin and eosin.)

at the time of the first reoperation in patient 1, probably because the collagen had not been completely reabsorbed and replaced by fibrous tissue.

Although good results have been reported when the Microvel Hemashield prosthesis is used in the high-pressure arterial system [1, 7, 8], very little is known experimentally or clinically about its use in the low-pressure pulmonary system. We emphasize the need for further investigation of the behavior of the Microvel Hemashield or indeed any other collagen-coated conduit when it is implanted in a low-pressure system before clinical use of such conduits is extended.

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Spontaneous Hemothorax in a Patient With Hereditary Multiple Exostoses

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A case of spontaneous hemothorax in a 7-year-old child secondary to erosion of the diaphragm by an exostosis coming from the left sixth rib is reported. This rare case of hemothorax with hereditary multiple exostoses is made even rarer by the concomitant perforation of the diaphragm.

(Ann Thorac Surg 1989;48:717–8)

Hereditary multiple exostoses is an autosomal dominant condition characterized by multiple exostoses usually in the long bones, with the scapula, pelvis, and ribs less often involved [1]. The exostoses are usually well tolerated, and patients generally live a normal life with only mild to moderate shortness of stature because of some growth retardation of the long bones. Malignant transformation to chondrosarcoma is a well-known but rare complication [2]. All other problems are the result of mechanical interference with neighboring anatomical structures such as the life-threatening problem we report here.

A 7-year-old boy known to have multiple exostoses with a positive family history was admitted to our hospital on December 7, 1988, with dyspnea of rapid onset that had been present for about 12 hours. For the four days before his admission, he had been taking antibiotics for sinusitis. When examined in the emergency department, he had pain in the left side of his chest and shortness of breath. Roentgenograms showed a pleural effusion that occupied approximately 50% of the left thoracic cavity. Some abnormalities of the second, third, and sixth ribs on the left were also noted. A pleural tap performed at this time showed noncoagulated blood in the thoracic cavity. With a thoracic drain, 400 mL of blood was withdrawn. No abnormal cells or bacteria were evident in the aspiration. Tests of coagulation were normal. Further radiological investigation showed other osteochondromas in the right second rib, on the scapula, in the left humerus, and in the spinal process of the fifth lumbar vertebra.

Family history revealed multiple exostoses in one of his brothers, his father, and two of his paternal aunts. In the 48 hours after installation of the thoracic drain, complete atelectasis of the left lung developed, and the patient continued to have light drainage. Thoracic computed tomographic scan performed on December 9, 1988, showed the exostosis from the sixth left rib anteriorly with a long bony spicule pointing directly toward the lung parenchyma (Fig 1).

The persistent drainage combined with the computed tomographic findings was considered indication for thoracotomy. At operation, we found a long bony spicule proceeding anteriorly from the left rib and eroding the diaphragm. The affected diaphragm was inflamed and showed a laceration. The part of the rib with the exostosis was resected. Manual exploration of the thoracic cavity showed several other exostoses on the second and third ribs, but they had smooth surfaces.

In the postoperative period atelectasis developed, again requiring bronchoscopies and intensive respiratory therapy, but the patient finally recovered completely, had normal roentgenograms, and was discharged 2 weeks after admission.

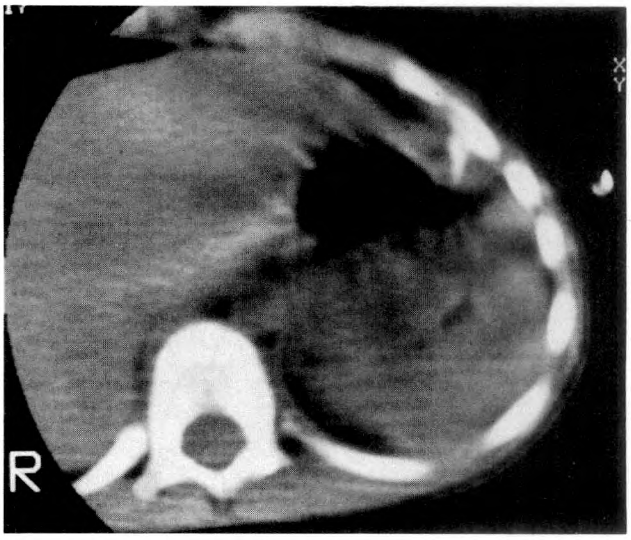

Fig 1. Computed tomographic scan shows exostosis from the sixth left rib.

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Comment

Review of the literature showed 3 other cases of hemothorax caused by exostoses. In the 2 patients of Proper and associates [3] and in the 1 patient reported by Balatskii and colleagues [4], the cause of the hemothorax was believed to be an irritation of the pleura by the exostosis. Only 1 of these 3 patients needed surgical treatment for persistent hemorrhage, as in our case. In our patient, the hemorrhage issued directly from the diaphragm, which had been eroded by the rather pointed bony exostosis.

This case of a spontaneous hemothorax caused by an exostosis perforating the diaphragm is rare. Exostosis perforating the diaphragm could be a life-threatening situation, and any patient with this condition should be watched closely. When an intrathoracic exostosis is directed toward an intrathoracic structure, serious consideration should be given to prophylactic surgical removal.

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Ventricular Pseudoaneurysm Associated With Cardiopulmonary Resuscitation 6 Weeks After Mitral Valve Replacement

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Trauma to the heart and mediastinum is associated with external cardiac massage. A patient had undergone a redo mitral valve replacement and experienced an uneventful postoperative course. During a visit to her physician 6 weeks after operation, she experienced ventricular fibrillation that required external cardiac massage and subsequent defibrillation. Postresuscitation evaluation revealed a posterior pseudoaneurysm of the ventricle. This was repaired via a transthoracic approach with the use of profound hypothermia.

(Ann Thorac Surg 1989;48:719–20)

Pseudoaneurysm of the ventricle is an uncommon but well-described entity that has been identified after myocardial infarction, cardiac operation, trauma, endocarditis, and cardiomyopathy [1]. Mitral valve replacement may predispose to a pseudoaneurysm, which can appear at any time, from intraoperatively to 6 years or more after operation [2, 3]. This report describes a 69-year-old woman who developed a pseudoaneurysm after cardiopulmonary resuscitation for ventricular fibrillation 6 weeks after her second mitral valve replacement.

A 69-year-old woman was admitted to Hartford Hospital after a cardiac arrest. The patient had had a history of rheumatic heart disease and had undergone a mitral valve replacement with a porcine valve in 1977. Ten years postoperatively, she had acute congestive heart failure secondary to acute mitral regurgitation. She underwent urgent redo mitral valve replacement with a 35-mm bioprosthesis and experienced an uneventful postoperative course. Six weeks after operation, she sustained a witnessed cardiac arrest as she left the office of her family physician. Cardiopulmonary resuscitation was initiated, and ventricular fibrillation was identified on electrocardiogram. Defibrillation restored her usual rhythm of atrial fibrillation. Intravenous administration of lidocaine was started, and she was admitted to the coronary care unit. Serial electrocardiograms and cardiac enzyme tests did not show evidence of myocardial damage.

The patient complained of severe upper back pain and shoulder pain radiating to her left arm. This resolved with administration of morphine, but her symptoms recurred approximately six hours later. An echocardiogram suggested the presence of a pseudoaneurysm posteriorly. Cardiac angiography confirmed the diagnosis of a pseudoaneurysm of the left ventricle. There was no mitral regurgitation, good left ventricular function was observed, and no clinically significant coronary artery disease was identified.

Repair of the pseudoaneurysm was undertaken via a left transthoracic approach to avoid the dense pericardial adhesions from her recent operation. Femorofemoral bypass was initiated, and the pulmonary artery was cannulated to accomplish total bypass. Hypothermia was induced through cardiopulmonary bypass. Ice bags were placed at the sides of the head, and Pentothal was administered at 40 mg/kg of body weight. At 19°C, circulatory arrest was induced. No abnormalities of the mitral valve were identified through the left atrium. A pseudoaneurysm of the ventricle was identified just under the atrial ventricular groove posteriorly, arising between the posterior descending branch and left ventricular branch of the right coronary artery. The pseudoaneurysm was opened, and a 1-cm perforation of the left ventricle was located just below the valve suture line. No scar formation or evidence of chronicity was identified at the site. The hole was closed with a series of Prolene (Ethicon) sutures over Teflon pledgets. Cardiopulmonary bypass was reinstituted, and the patient's temperature was returned to normothermic levels. She was weaned from cardiopulmonary bypass without difficulty, and awakened six hours postoperatively with no apparent neurological sequelae. Her postoperative course was complicated by ventricular ectopy. Electrophysiological studies were performed, and her ventricular irritability was suppressed with flecainide. The remainder of her postoperative course was uneventful.

Comment

Mitral valve replacement may predispose to formation of ventricular pseudoaneurysm. Presumably, surgical manipulation of the mitral valve annulus may result in weakness of the ventricular wall. A partial rupture of the ventricular wall produces a pseudoaneurysm composed

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of epicardium and pericardial adhesions. Varied symptoms of ventricular pseudoaneurysm include chest pain and dyspnea, endocarditis, an abnormal left heart border on chest x-ray film, myocardial infarction, and a murmur of mitral regurgitation. Ventricular pseudoaneurysm also has been discovered as an unrelated finding at autopsy in patients who have undergone mitral valve replacement. Pseudoaneurysm precipitated by cardiopulmonary resuscitation in a patient who has undergone mitral valve replacement is rare. The second mitral valve replacement in this patient may have predisposed her to formation of a ventricular pseudoaneurysm. There was no evidence of ventricular pseudoaneurysm during her postoperative course, and she was enjoying a return to a normal activity level at the time of her cardiac arrest. Onset of symptoms after her resuscitation leads us to believe that the external chest compressions were directly related to the acute formation of her ventricular pseudoaneurysm. In patients who have undergone prosthetic mitral valve replacement and who are subsequently exposed to cardiopulmonary resuscitation or blunt trauma, ventricular pseudoaneurysm should be suspected.

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Coronary Artery Bypass in Idiopathic Thrombocytopenia Without Splenectomy

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A patient with coronary artery disease and idiopathic thrombocytopenia purpura underwent coronary artery bypass grafting without splenectomy. Our experience indicates that use of cardiopulmonary bypass in patients with idiopathic thrombocytopenic purpura does not invariably mandate splenectomy.

(Ann Thorac Surg 1989;48:721–2)

Cardiac operations requiring cardiopulmonary bypass (CPB) have been safely performed in patients with a variety of coagulation disorders, but reported experience in patients with idiopathic thrombocytopenic purpura is limited. We could find only 1 reported case in the English-language literature [1] and only a few cases in the Japanese literature [2]. None of the reports dealt with patients undergoing myocardial revascularization, and in each of these cases a prophylactic splenectomy was performed. We report our management of a patient with idiopathic thrombocytopenic purpura who underwent surgical revascularization without splenectomy.

A 61-year-old man had a 3-month history of angina pectoris and a positive exercise stress test. Coronary angiography revealed a total occlusion of the proximal left anterior descending coronary artery and a 75% stenosis of an obtuse marginal artery and first diagonal branch. The patient was admitted to the hospital for percutaneous transluminal coronary angioplasty.

The patient's past medical history included a diagnosis of chronic idiopathic thrombocytopenic purpura. The patient had spontaneous hematuria; the hematuria failed to resolve and his platelet count did not increase in response to steroid therapy. Physical examination was normal. Reevaluation of the patient's idiopathic thrombocytopenic purpura on admission included a peripheral blood smear with few platelets of normal morphology, a prolonged bleeding time, normal blood clotting time, and a bone marrow biopsy that demonstrated an increased number of megakaryocytes. The level of platelet-associated immunoglobulin G was increased.

Attempted percutaneous transluminal coronary angioplasty was aborted because the left anterior descending coronary artery lesion could not be traversed. The patient was referred for surgical revascularization. Exogenous platelet survival was evaluated preoperatively by administration of one type-specific platelet unit, which increased the platelet count from 68,000 to 84,000/mL. The count returned to 68,000/mL after 36 hours. A preoperative splenectomy was not performed.

The patient underwent a three-vessel coronary bypass procedure, receiving a left internal mammary artery graft to the left anterior descending coronary artery and reversed saphenous vein grafts to the diagonal and obtuse marginal branches. The total CPB time was 100 minutes; a membrane oxygenator was used. At the conclusion of CPB, the patient's intraoperative platelet count was 44,000/mL. Two units of platelets were administered during the procedure, and six hours postoperatively the platelet count was 150,000/mL. Postoperatively, the patient bled from the anatomical bed of the mammary artery and required surgical reexploration. During reexploration, he received 2 U of packed red blood cells and no platelets. Eighteen hours postoperatively, he received 1 U of platelets. The remainder of his postoperative course was uneventful. At discharge, the patient's platelet count was 110,000/mL; at 1 month after discharge, it was 67,000/mL. A stress test 6 months postoperatively showed no evidence of ischemia.

Comment

Idiopathic thrombocytopenic purpura is a disease characterized by a decreased number of circulating platelets (often fewer than 50,000/mL), which results from a shortened life span of blood elements. The most notable clinical manifestations are spontaneous hemorrhage and excessive posttraumatic bleeding. Splenectomy has been shown to increase platelet counts and alleviate symptoms even in patients who do not respond to medical therapy. It would be logical, therefore, to perform splenectomy prophylactically in patients about to undergo CPB. The reported experience with CPB in these patients is limited. Three cases have been reported, two involving mitral valve replacement and one involving primary repair of an atrial septal defect [1, 3, 4]. In each case, a splenectomy was performed prophylactically before the cardiac operation. No complications were reported in these cases. To our knowledge, this case is the first reported involving coronary artery bypass grafting and the first involving CPB without the use of splenectomy. We believed that the

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patient would tolerate this procedure because we had demonstrated that administration of exogenous platelets would increase the platelet count for at least 24 hours. This was confirmed even after CPB because administration of two six-packs of platelets increased the platelet count from 68,000 to 150,000/mL.

Results in our patient show that CPB and coronary bypass grafting may be performed in patients with idiopathic thrombocytopenic purpura. We suggest transfusion of platelets after termination of CPB as an alternative to prophylactic splenectomy in such patients. This alternative is particularly appealing in patients with serious coronary artery disease, in whom performance of splenectomy is associated with an increased risk of intraoperative cardiac morbidity [5].

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Candida Endocarditis With Saddle Embolism: A Successful Surgical Intervention

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A 43-year-old woman who had *Candida* endocarditis with saddle embolism underwent mitral valve replacement and resection of infected aorta. Only 7 persons with fungal endocarditis with saddle embolism have been reported in the literature, and all of them died. We report a case of successful treatment of this lethal complication of *Candida* endocarditis.

(Ann Thorac Surg 1989;48:723–4)

Candida endocarditis is a highly lethal disease. In particular, the prognosis of *Candida* endocarditis with embolism is extremely grave. We report a case of successful treatment of *Candida* endocarditis associated with saddle embolism in which mitral valve replacement and resection of infected aorta provided a satisfactory result.

A 43-year-old woman had undergone open mitral commissurotomy together with tricuspid annuloplasty for mitral stenosis and tricuspid regurgitation in 1973. She had been well for 11 years. On August 25, 1983, she suddenly had a syncopal attack and right hemiparesis due to cerebral embolism. Her symptoms improved during 2 weeks of hospitalization. She was admitted to Osaka University Hospital on February 1, 1984, complaining of exertional dyspnea and palpitation.

On physical examination at admission to the Osaka University Hospital, she was pale and appeared ill. She had no cyanosis, petechiae, or Osler's nodes. The neck veins were not distended. A grade 3/6 harsh holosystolic murmur and diastolic rumble were audible at the apex. Her temperature was 37.7°C. Her heart rate was 68 beats/min, and her blood pressure was 116/68 mm Hg. The liver was enlarged 5 cm below the right costal margin. The right femoral pulse and the pulses below the bilateral femoral arteries were not palpable. Laboratory examination showed a white blood cell count of 3,100/μL with normal differentiation. The hemoglobin value was 7.8 g/100 mL. The erythrocyte sedimentation rate was 27 mm/h. The urine output was normal. Cardiac catheterization showed mitral stenosis with mild regurgitation, mild aortic regurgitation, and moderate tricuspid regurgitation. Aortography revealed saddle embolism at the bifurcation of the aorta.

The patient's temperature ranged from 38° to 40°C every day after hospitalization. Several blood cultures grew *Candida albicans*. Echocardiogram revealed a large vegetation on the mitral valve (Fig 1). The patient received 20 mg of amphotericin B daily, but her high fever continued.

On February 14, five days after administration of amphotericin B was started, she underwent operation. The mitral valve had fungal vegetations and was removed; a 27-mm Björk-Shiley prosthetic valve was implanted. Tricuspid annuloplasty was added for tricuspid regurgitation. There were no thrombi in the left atrium.

Histological section showed vegetations with numerous yeast colonies and hyphae (Fig 2). Administration of 30 mg of amphotericin B daily was continued after operation, but one of four blood cultures was positive for *Candida*.

The operation for the saddle embolism was performed on February 28, after the patient was well recovered. The distal abdominal aorta and bilateral iliac arteries were resected. A Sauvage Y-shaped graft, which had been immersed in glue containing amphotericin B, was implanted between the abdominal aorta and bilateral iliac arteries. The resected bifurcation was filled with a brittle gray-white embolic material (Fig 3). Microscopic examination showed granulomatous aortitis with organized thrombus, and the culture of this resected material grew C *albicans*.

All blood cultures for fungus were negative after the second operation, and her postoperative course was uneventful. Intensive medical treatment for candidiasis was continued with intravenous amphotericin B, 30 mg daily. A total dose of 1.3 g of amphotericin B was administered over a 2-month period. Oral 5-fluorocytosine was instituted in a dose of 4 g daily.

She was discharged on May 12, receiving digitalis, diuretics, warfarin, and 5-fluorocytosine. Administration of 5-fluorocytosine was continued for 6 months after her discharge. She has been doing well for 4.5 years and has normal hematologic, renal, and hepatic function.

Comment

Candida endocarditis is a highly lethal disease. The mortality of fungal endocarditis with medical treatment is greater than 80% [1]. In 1961, Kay and associates [2] reported successful excision of a tricuspid valve infected with C *albicans* and advocated more aggressive surgical management of active *Candida* endocarditis. They also

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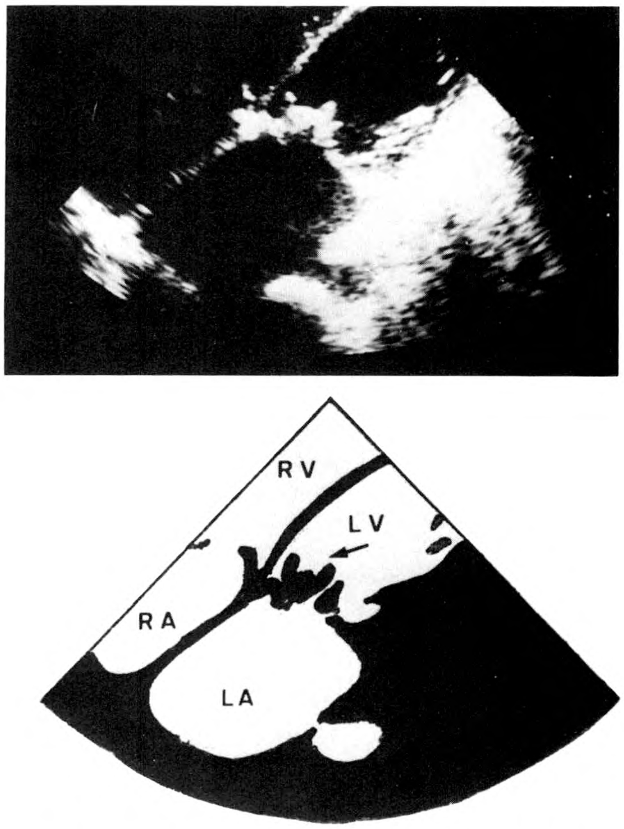

Fig 1. Two-dimensional, four-chamber, apical view shows shaggy mass (arrow) *on the mitral valve* (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

emphasized that surgical therapy in combination with antifungal drugs was mandatory in management of *Candida* endocarditis. In the subsequent literature, 55% to 80% of patients who were treated with combined surgical and medical therapy have survived. Therefore, the only hope for survival is aggressive surgical intervention in combination with intensive medical therapy.

Embolism occurs in 33% to 75% of the patients with *Candida* endocarditis and is apt to occur in the large arteries [3, 4]. However, saddle embolism is rare. Only 7 persons with fungal endocarditis and saddle embolism have been reported in the previous literature, and all of them died [1, 3–8]. Five of these 7 patients underwent embolectomy only. No patient underwent both resection of the infected cardiac valve and resection of aortic bifurcation. We report a successful case of a patient who was infected by *Candida* with endocarditis and saddle embolism. We believe that our success was due to removal of all the *Candida* foci.

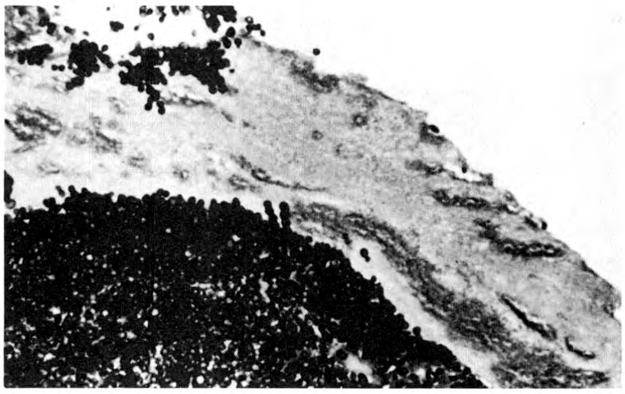

Fig 2. Histological section of vegetations on the mitral valve shows yeast colonies and hyphae. (Periodic acid-Schiff stain; ×400 before 53% reduction.)

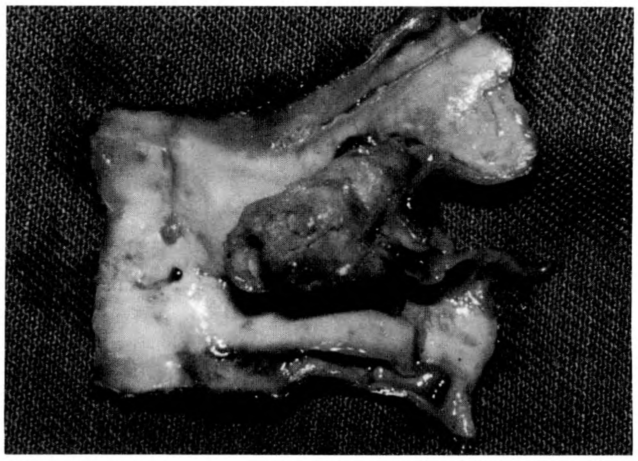

Fig 3. A large infectious thrombus occluded bifurcation of the aorta.

If possible, simultaneous operations for *Candida* endocarditis and embolism may be better for treatment of this highly lethal disease. However, we performed a two-stage operation because we believed a simultaneous procedure might worsen the condition of our debilitated patient.

The literature describes many relapses after management of *Candida* endocarditis. However, after 4.5 years, we believe that our patient is cured.

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Modified Danielson Technique for Recurrent Aortic Valve Endocarditis

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Recurrent aortic valve endocarditis is frequently associated with paravalvular ring abscess that destroys the annulus. In occasional cases, the degree of necrosis, destruction of the annulus, and the presence of intramyocardial abscesses make it impossible to seat a new prosthesis. Danielson reported initial success in treating such patients by translocating the aortic valve to the ascending aorta and placing vein grafts to the coronary arteries. We recently treated a 17-year-old patient suffering recurrent aortic valve endocarditis using Danielson's technique, which we modified by implanting the left main coronary artery directly into the conduit. We believe that in young patients this modification can offer a beneficial alternative.

(Ann Thorac Surg 1989;48:725–7)

Recurrent aortic valve endocarditis after valvular replacement remains a surgical challenge. Reoperations are often required to treat such patients. Aortic root abscess resulting from ongoing endocarditis, as well as multiple procedures, often leads to annular destruction. Aortic valve replacement or reinsertion in these cases becomes hazardous and, whenever feasible, often results in recurrent endocarditis, highly lethal in such situations.

Danielson and colleagues [1] described an original approach to manage such patients. They suggested translocating the aortic valve on the ascending aorta just above the coronary ostia; however, the counterpart of this procedure is that bypass grafts to the coronary arteries are necessary. Reitz and co-workers [2] used a valved conduit, which they interposed on the ascending aorta. We recently modified Danielson's technique by reimplanting the left main coronary artery (LMCA) directly into the conduit instead of using a venous graft in a 17-year-old patient with recurrent native aortic endocarditis owing to *Enterococcus*, operated on twice in a 2-month period. The first operation consisted of an aortic valve replacement with a No. 21 Carpentier-Edwards bioprosthesis as well as an autologous pericardial patch to close an aortic root abscess. Twenty-eight days later, a massive aortic regurgitation occurred. The second operation showed a circumferential disinsertion of the prosthesis and a massive destruction of the annulus and the aortic root. A Dacron patch was sutured to reconstitute the aortic root, and a St. Jude medical valve (No. 21) was seated with Teflon pledgets. Three weeks later, massive aortic incompetence recurred, leading to the third operation now described.

Technique

Cardiopulmonary bypass was instituted between the aorta and both venae cavae. Myocardial protection was undertaken by right atrial retrograde cardioplegia and topical cooling. The previous aortotomy was reopened, and the lesions were analyzed. In this patient at the third reoperation in a 2-month period, anatomical replacement of the aortic valve clearly was not reasonable owing to multiple vegetations and abscesses as well as a disinsertion of the existing prosthesis for half of the circumference. After a careful debridement of all necrotic tissues, a Danielson-type operation appeared mandatory. The aorta was transected 1.5 cm above the right coronary ostia, which was oversewn. A 3-cm-long aortic segment was then resected. The origin of the LMCA was tailored together with an aortic cuff, and the LMCA was dissected distally (Fig 1A) and mobilized up to its bifurcation. A valved conduit with an Ultracor prosthesis was then interposed. The LMCA was directly reimplanted on the posterolateral aspect of the prosthesis just above the valve (Fig 1B). An aorta–right coronary artery graft was then performed using a 3-cm saphenous vein graft between the anterior aspect of the tube and the proximal portion of the right coronary artery (Fig 1C). The postoperative course was uneventful. The patient was discharged on the 25th postoperative day, afebrile and with no aortic regurgitation. At 6 months, he is still asymptomatic with no fever or heart murmur. Control angiograms performed at 6 months show a patent LMCA and no paravalvular leak (Fig 2).

Comment

Iterative aortic valve endocarditis is highly lethal and constitutes a surgical challenge [1, 2]. The technique described by Danielson and colleagues [1] is a useful option for such patients. It has simplified the surgical procedure by allowing the surgeon to work outside the infected site and thus decreased the rate of paravalvular

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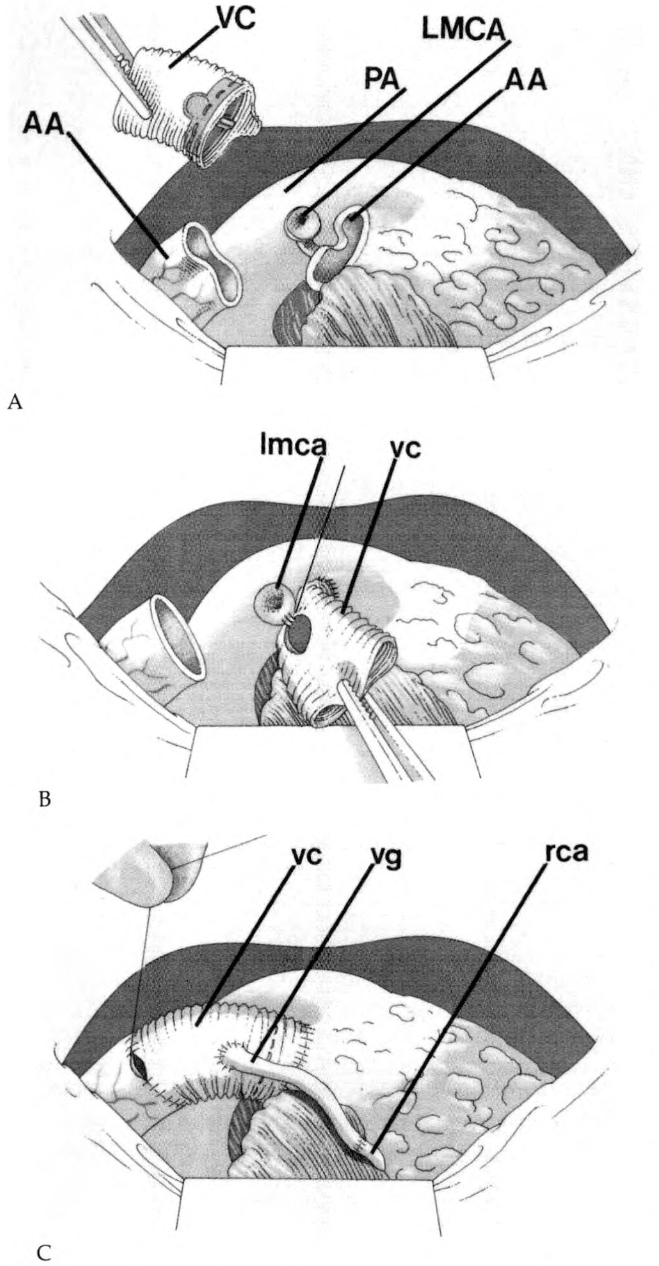

Fig 1. (A) A 3-cm segment of the aorta was resected and the left main coronary artery (LMCA *or* lmca) *was dissected. (B) The LMCA was reimplanted on the posterior aspect of the conduit. (C) A 3-cm vein graft was interposed between the conduit and the proximal right coronary artery* (rca). (AA = *ascending aorta;* PA = *pulmonary artery;* VC *or* vc = *valved conduit;* vg = *vein graft.)*

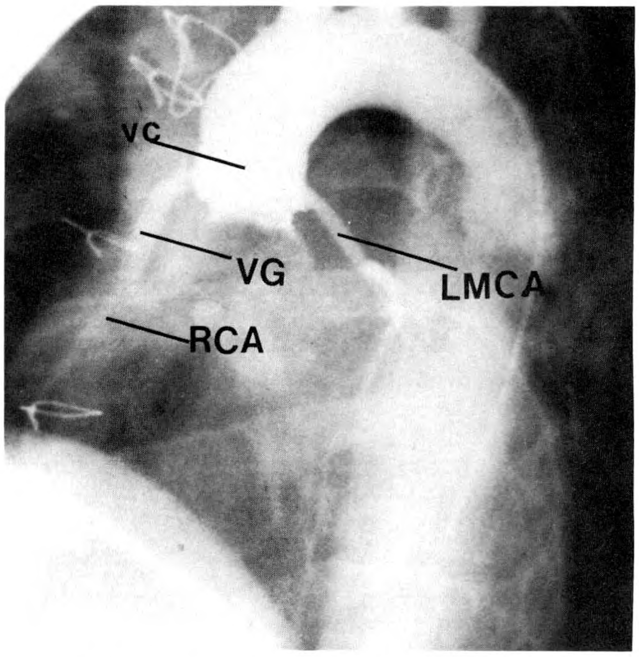

Fig 2. Control angiogram at 6 months: The left main coronary artery (LMCA) *is patent. The vein graft to the right coronary artery* (RCA) *is evident, and there is no paravalvular leak.* (vc = *valved conduit;* vg = *vein graft.)*

leaks and reoperations. However, in young patients with aortic valve endocarditis, use of vein grafts to bypass the coronary arteries appears unfavorable now that the fate of these latter conduits is better elucidated [3]. This was the main rationale for our technique. Moreover, in select cases it may be possible in a similar manner to dissect the right coronary artery free and to reimplant it directly into the conduit and thus not use vein grafts at all. In our case, this was judged impossible due to the tight adhesions at this site. Nonetheless, we believe this technical modification offers to the excellent procedure described by Danielson and associates a certain amelioration for the long term.

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INVITED COMMENTARY

Dr Dreyfus and co-authors are to be commended for their salvage of a patient who had recurrent aortic root endocarditis with multiple abscesses. They employed a modification of the aortic valve translocation procedure, an operation that was initially developed to salvage a similar desperate situation in a young patient who, after aortic valve replacement for staphylococcal endocarditis, developed reinfection, extensive annular abscesses, and dehiscence of the prosthesis. Translocation of the valve prosthesis to the ascending aorta with restoration of supravalvular coronary blood flow allowed complete healing of the aortic root. There is no question that select patients with aortic root infections caused by especially virulent organisms have survived and been cured of their infections by translocation of the aortic valve, when other procedures have failed. The variation by Dreyfus and associates has the advantage of eliminating the need for one saphenous vein graft. However, it introduces an additional foreign body (Dacron graft) in proximity to the infected area, which theoretically might increase the possibility of reinfection. Additional experience with this modification is warranted.

The concern for late vein graft deterioration is a valid one, although this complication may be less likely to occur in younger patients than in the typical older patient undergoing myocardial revascularization for advanced atherosclerosis. The aortic valve translocation operation does not preclude eventual return of the aortic valve prosthesis to the subcoronary position once the infection has healed, should this be necessary because of late deterioration of the vein grafts.

Our experience with aortic valve translocation now numbers 8 patients. Fortunately, it is not an operation that must be done frequently. All but 2 patients had prosthetic valve endocarditis, and the ages ranged from 15 to 76 years. For 1 patient, it was the third aortic valve replacement, and for another, the fourth valve replacement. There were two intraoperative deaths and one death at 2 weeks from left ventricular failure. These deaths from cardiac failure emphasize the necessity of considering this operation before the patient is in terminal heart failure. The remainder of the patients survived operation without recurrence of infection.

There is currently another option for surgical management of patients with prosthetic valve endocarditis and aortic root abscesses who have failed more conservative procedures. This method includes thorough debridement of the aortic root and placement of an antibiotic-preserved or cryopreserved homograft aorta with integral aortic valve. The lower end of the aorta is sewn to the subaortic tissues or mitral valve annulus. The coronary arteries are implanted end-to-side into the aorta above the valve, and the distal aorta is anastomosed end-to-end intraluminally to the ascending aorta. We have employed this technique, using cryopreserved aortic homografts, both for aortic root abscesses in 7 patients and for correction of late endocarditis in 3 patients who had undergone a Konno procedure for tunnel subaortic stenosis. In the latter 3 patients, the anterior leaflet of the mitral valve was used to close the ventricular septal defect. These 3 patients and 5 of the other 7 survived operation and were free of reinfection. These early experiences of our group and those of others suggest that the homograft aorta is more resistant to reinfection than other prosthetic materials. Further experience is needed to determine the relative merits of the homograft aorta versus translocation of the aortic valve for treatment of complicated types of aortic root endocarditis.

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Coronary Artery Bypass Grafting in Patients With Calcified Ascending Aorta: Aortic No-Touch Technique

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To perform coronary artery bypass grafting safely for patients with calcified ascending aorta, an "aortic no-touch technique," which consisted of (1) maximal utilization of in situ arterial grafts, (2) fibrillatory arrest without aortic cross-clamp, (3) left ventricular venting through the right superior pulmonary vein, and (4) femoral artery perfusion, was attempted in 3 patients. All were men, aged 64, 70, and 56 years, respectively, with triple-vessel disease with severe atherosclerotic lesion in the ascending aorta. Bilateral internal mammary arteries and the right gastroepiploic artery were used in all patients. All patients survived without evidence of perioperative myocardial infarction or cerebrovascular accident.

(Ann Thorac Surg 1989;48:728–30)

Atherosclerotic ascending aorta has been known as a critical risk factor in performing coronary artery bypass grafting because of difficulty in proximal anastomosis of the saphenous vein graft and the risk of aortic dissection and cerebral embolism due to intimal crush by aortic clamp [1, 2]. To avoid these unfavorable complications, coronary artery bypass grafting with "aortic no-touch technique" was successfully performed in 3 patients with triple-vessel disease and calcified ascending aorta.

Material and Methods

All 3 patients were men, aged 64, 70, and 56 years, respectively. Coronary arteriogram and left ventriculogram showed severe triple-vessel disease with minimal impairment of ventricular contraction in these patients. Calcification was noted in the ascending aorta at preoperative cineangiogram in 2 patients and was confirmed by intraoperative palpation in all patients.

Surgical procedure consisted of (1) maximal utilization of in situ arterial grafts (ie, bilateral internal mammary arteries and right gastroepiploic artery) to bypass at least three major branches of the coronary artery, (2) fibrillatory arrest without aortic cross-clamp with topical cooling and rectal temperature at 25°C, (3) left ventricular venting through the right superior pulmonary vein, and (4) femoral artery perfusion (Fig 1). Perfusion pressure was maintained around 80 mm Hg as Akins [3] recommended. The internal mammary artery and the gastroepiploic artery were taken down with techniques previously described [4] and anastomosed to the coronary artery with continuous suture technique using single 8-0 Prolene (Ethicon) suture. Each of the coronary arteries was snared with a 4-0 Prolene suture proximally for local isolation to avoid bleeding at the time of anastomosis.

All 3 patients received triple coronary artery bypass with the right internal mammary artery to the left anterior

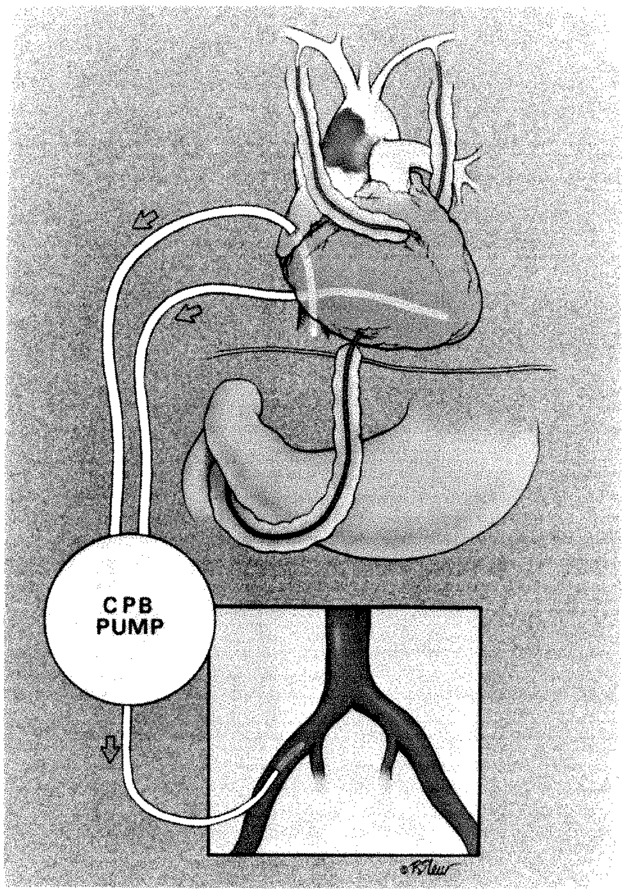

Fig 1. Scheme of aortic no-touch technique: maximal utilization of in situ arterial grafts using bilateral internal mammary and gastroepiploic arteries, fibrillatory arrest without aortic clamp, left ventricular vent, and femoral artery perfusion. (CPB = *cardiopulmonary bypass.)*

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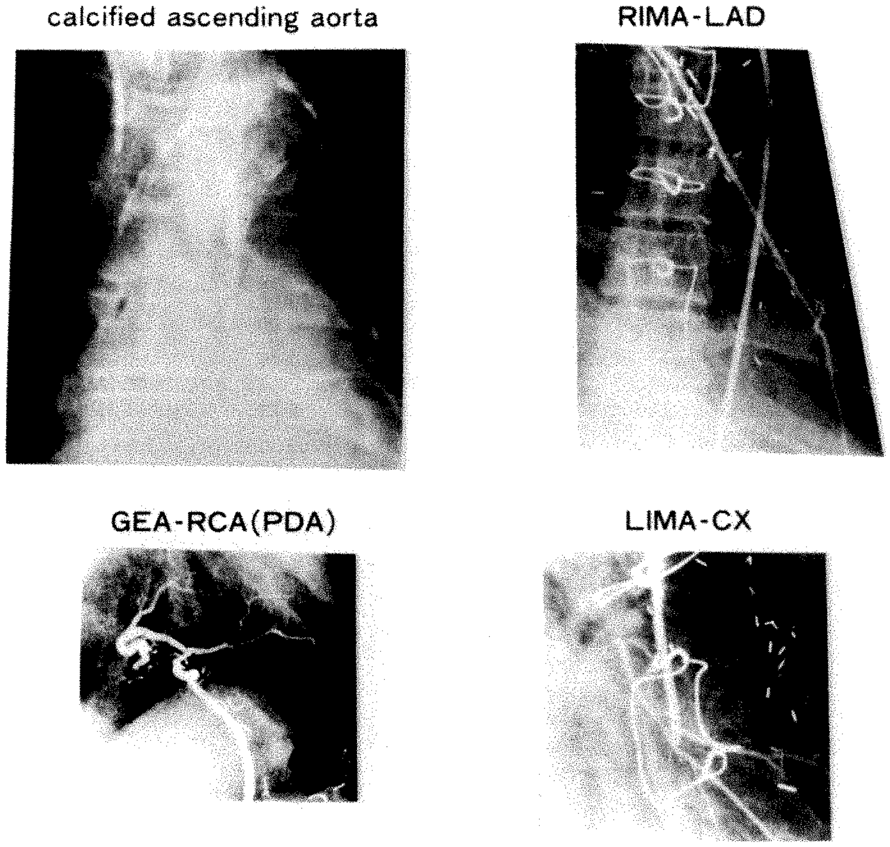

Fig 2. Calcification of ascending aorta (left top) and postoperative angiograms demonstrating patent right internal mammary artery (RIMA) anastomosed to the left anterior descending artery (LAD) (right top), left internal mammary artery (LIMA) to the circumflex artery (CX) (right bottom), and the right gastroepiploic artery (GEA) to the right coronary artery (RCA) (left bottom) in a 64-year-old man.

descending artery, the left internal mammary artery to the circumflex artery, and the right gastroepiploic artery to the right coronary artery. Mean duration of ventricular fibrillation and cardiopulmonary bypass was 54.7 minutes and 116.8 minutes, respectively.

Results

All patients survived without evidence of perioperative myocardial infarction. There were no signs of cerebrovascular accident after operation. Postoperative angiography was performed in 2 patients at 4 weeks after operation, and all grafts were patent. Figure 2 shows calcification of the ascending aorta and a postoperative angiogram in a 64-year-old man. Relief of angina was obtained in all patients.

Comment

Atherosclerotic ascending aorta is a troublesome complication in performing coronary artery bypass grafting because of the (1) difficulty of proximal anastomosis of the saphenous vein graft due to thickening of the aortic wall, (2) potential risk of aortic dissection due to laceration of intima by the aortic clamp, and (3) high incidence of cerebrovascular accident due to embolization of crushed debris damaged by the aortic clamp. To avoid these unfavorable complications, Mills [5] described a technique using bilateral internal mammary artery in situ grafts in conjunction with saphenous vein grafts as the Y graft without clamping the aorta. Landymore and colleagues [1] reported a technique of arch cannulation and intermittent clamping of arch vessels to prevent cerebral embolism in this situation. Holland and Hieb [2] described the successful outcome of 3 such patients after anastomosis of the proximal saphenous vein graft to the root of the carotid artery in combination with an internal mammary artery graft. I recommended maximum utilization of in situ arterial grafts, by which bypassing even four or five coronary arteries is possible by using bilateral internal mammary arteries and right gastroepiploic artery with a sequential grafting technique. The suitability of the right gastroepiploic artery as a coronary bypass graft was reported previously [4]. Reliability of fibrillatory arrest in terms of myocardial protection has been proved by Akins [3]. When femoral arteries are also calcified and stenosed, the soft part of the aortic arch is the best site of cannulation, as Landymore and associates [1] suggested.

In conclusion, coronary artery bypass grafting with aortic no-touch technique using internal mammary artery and gastroepiploic artery grafts is safe and effective for ischemic heart patients with atherosclerotic ascending aorta.

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REVIEW OF RECENT BOOKS

Aortic Surgery
Edited by John J. Bergan and James T. Yao
Philadelphia, W.B. Saunders, 1988
574 pp, illustrated, $99.00

Reviewed by A. M. Graham, MD

Once more Drs Bergan and Yao have put together a very impressive list of authors, in their multiauthored textbook entitled *Aortic Surgery*. Their annual contribution to the vascular literature has always produced textbooks of high quality, and this book follows in that tradition. As mentioned, the book has many authors, although there is a strong flavor of the Northwestern University experience throughout the book as a number of chapters are authored by this group of vascular experts. The book begins with a fascinating chapter on the surgery of the aorta by Dr Wylie Barker. This delightful and enlightening start to the textbook reminds us of how much courage and intelligence our founders in vascular surgery had, and careful reading is a must for all vascular surgeons. The second part of the textbook deals with basic considerations with respect to the aorta and operations on it. The group from the University of Chicago led by Dr Christopher Zarins nicely summarizes years of research by this center in the pathogenesis of atherosclerosis.

Before a number of chapters on the multiple problems with the aorta and operation on it, there is an informative section regarding preparation of the patients for aortic surgery. This again is a good review of all the techniques that are available to the vascular surgeon and of the physiology that is involved in dealing with the risk factors that are important in maintaining high standards of care in aortic surgery. After this, the book goes on to describe multiple problems in the aorta; some of them, unfortunately, are dealt with in a superficial manner. Some chapters that may seem important to many are of such brevity that the reader certainly learns very little from them. This of course is a problem that occurs with multiauthored textbooks and is something that must be dealt with at an editorial level. It certainly detracts from the textbook when there are a number of chapters that do not match the standards of the rest of the book.

However, certain chapters do stand out, especially the chapter on Takayasu's disease and Dr Bergan's presentation on the variations of the Leriche syndrome. Throughout the book are chapters by the true experts in the field of aortic surgery, and the group from the University of California in San Francisco does not let us down with their chapter on pararenal aneurysms. Presentations of the use of the supraceliac aorta as inflow for distal bypass grafts and the retroperitoneal approach to aortic reconstruction are timely and informative chapters.

In summary, this book is an important addition to the library of all vascular surgeons. It is an especially good review textbook on aortic reconstruction for the Fellow training in vascular surgery and preparing for his or her Vascular Fellowship. We expect nothing less than this type of excellence from Dr Bergan and Dr Yao. I would highly recommend this book.

Montreal, Quebec, Canada

Construction of an Aortic Homograft Conduit for Right Ventricle to Pulmonary Artery Continuity

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Over the past decade the aortic homograft has become the extracardiac conduit of choice for repair of many congenital cardiac defects. Anastomosis of the homograft annulus to the right ventricle may distort the valve and render it incompetent. Prosthetic tube grafts have been used to bridge the right ventricle and distal homograft. We propose and have used successfully a method for establishing right ventricle to pulmonary artery continuity that is free of prosthetic materials, is technically simple, and places the homograft valve in the hilum, away from the distorting right ventricle free wall.

(Ann Thorac Surg 1989;48:731–2)

This patient was a 6-year-old girl with truncus arteriosus, type I. She underwent repair at age 6 months with a 15-mm porcine-valved Dacron right ventricle (RV) to pulmonary artery (PA) conduit. Exercise intolerance and dyspnea developed. She had a harsh grade 5/6 systolic murmur heard best at the left upper sternal border. Chest roentgenogram showed calcification of the porcine valve. Cardiac catheterization revealed conduit obstruction and valve stenosis with a 60-mm Hg gradient across the porcine valve. She underwent replacement of the Dacron and porcine valve conduit with a 21-mm homograft aortic conduit; the valve was placed at the bifurcation of the main pulmonary artery (Figs 1, 2). The Dacron had a thick layer of neointima that was firmly attached. The porcine valve was severely calcified and stenotic. The patient made an uneventful recovery and is asymptomatic.

Technique

When replacement of an RV–PA conduit is indicated, we resect the old conduit completely from the RV and from the PA. All Dacron is removed. The homograft is prepared for the PA anastomosis. A straight transverse cut across the graft just above the sinotubular junction is done. If the anastomosis needs to be carried to one side of the PA bifurcation, the homograft is tailored to the PA anatomy. The septal myocardial tissue and mitral tissue are debrided before placement. The remainder of the aorta is then reversed and the aortic arch end cut obliquely to match the ventriculotomy. All anastomoses are done with 5-0 polypropylene continuous suture. The proximal conduit is then trimmed to fit the distal portion end-to-end and the anastomosis is completed. The conduit is soft and usually lies toward the left so that compression from the sternum is unusual.

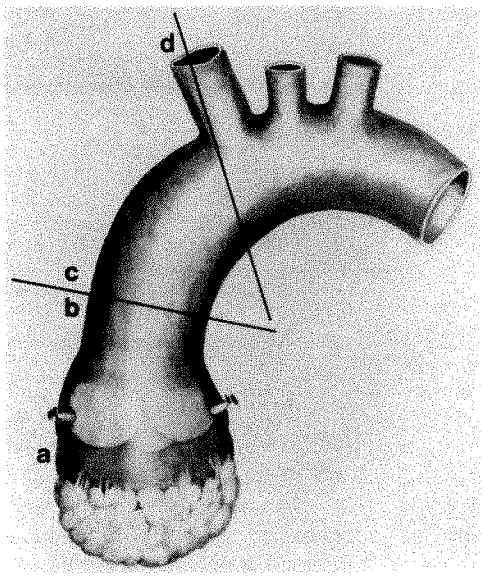

Fig 1. The homograft of aortic valve and ascending aorta is divided first at the sinotubular junction and again in the transverse section to accommodate the ventriculotomy.

Comment

The repair of complex congenital cardiac defects has been greatly aided by use of the extracardiac conduit. Dacron has been popular in the past, but pseudointimal formation as described by Agarwal and associates [1] can cause obstruction and right ventricular failure. Another cause of obstruction can be calcific degeneration of the porcine valve [2]. An aortic homograft with integral valve was used by Ross and Somerville [3] in 1966 for repair of tetralogy of Fallot. In the past decade this type of graft has become the extracardiac conduit of choice. Valve-containing conduits, which prevent regurgitation, allow the right ventricle to compensate for acute changes in pulmonary vascular resistance postoperatively better than do nonvalved conduits. The homograft does undergo calcification in the long term, but the antibiotic-sterilized homografts are unlikely to become obstructed [4].

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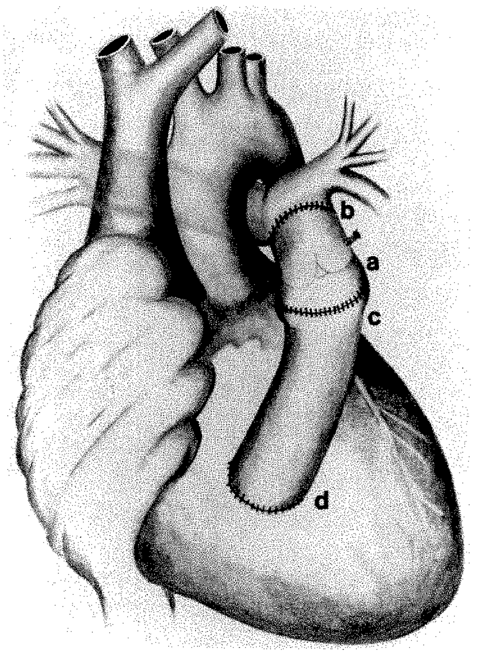

Fig 2. The homograft has been anastomosed first to the pulmonary artery and then to the right ventricle. The final anastomosis joins the two sections. The letters *correspond to the same sections pictured in Figure 1.*

We feel it is important to place the homograft valve in a distal position next to the PA to prevent distortion of the valve [4, 5]. Others have placed the homograft valve into a distal position using Dacron or autogenous pericardium to bridge the space between the right ventricle free wall and the homograft valve [5, 6]. In using homograft material to bridge the space, the possibilities of fibrous peel obstruction from Dacron [7, 8] and late aneurysmal formation from pericardium may be eliminated. Fibrous peel or pseudointimal growth has not been recognized as an important sequela of long-term homograft use [6]. The distal anastomosis should be done first, as it is the more difficult. The homograft should be cut just above the sinotubular junction to match the distal PA. All anastomoses are with a nonabsorbable monofilament suture. Excess homograft septal and mitral tissue should be debrided before starting the anastomosis.

The remaining portion of the homograft is then used to bridge the RV to homograft valve. This is usually best accomplished by reversing the conduit and sewing the properly tapered transverse aortic section to the RV. The final step is to properly match the length of the conduit by trimming and then to anastomose the two homograft segments.

By securing the two portions of homograft to the relatively immobile patient structure, the final anastomosis between the two homograft segments can be accomplished efficiently.

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Implantation Techniques for the Hemopump

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and Vladimir Velebit, MD

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Techniques for implantation of the Hemopump, an intraarterial, axial-flow circulatory assist device, are described. The Hemopump, which is currently undergoing clinical investigation, has been used successfully to treat patients experiencing profound left ventricular failure in a variety of clinical situations, including postcardiotomy shock, acute myocardial infarction, cardiac allograft rejection, and cardiac allograft failure.

(Ann Thorac Surg 1989;48:733–5)

The Hemopump, an axial-flow circulatory support device recently developed by Nimbus Medical (Rancho Cordova, CA), is currently undergoing clinical investigation [1]. Clinical evaluation of the device began at the Texas Heart Institute in April 1988 [2]. Patients in the study had experienced (1) cardiogenic shock after myocardial infarction, (2) inability to be weaned from cardiopulmonary bypass during operation despite conventional pharmacological and intraaortic balloon pump support, (3) cardiac allograft rejection, or (4) allograft failure.

A variety of techniques for implanting the Hemopump have been developed. Some are new and others are adapted from techniques used for intraaortic balloon pump insertion. This report describes implantation techniques that we have found effective.

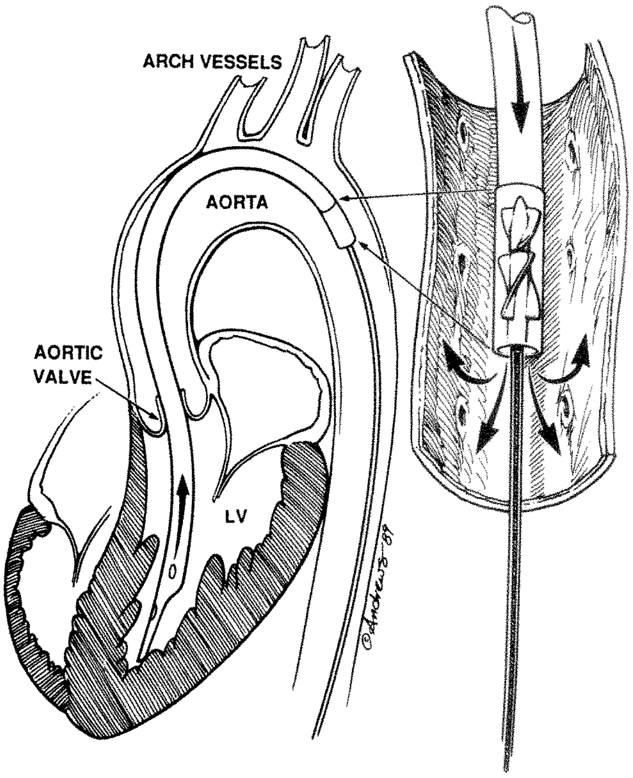

Fig 1. Position of Hemopump in left ventricle and aortic arch, with detail of the pump impeller. (LV = *left ventricle.)*

Material and Methods

The Hemopump consists of a pump assembly, a high-speed motor, and a drive console. The disposable pump assembly integrates all blood-contacting surfaces into a single system. Power to the axial-flow pump (located within the housing) is transmitted by a flexible drive shaft enclosed in a 9F polymeric sheath. The external end of the drive shaft is connected to an external reusable high-speed motor. When the device is activated, the pump impeller, which is capable of rotating at speeds up to 27,000 rpm, provides unidirectional nonpulsatile flow. The pump propels blood into the aorta from the left ventricle; the maximum flow rate approaches 4 L/min. Two types of cannulas (7-mm diameter), both made of silicone, have been used clinically with the Hemopump. A short cannula (10 cm) is designed for insertion into the ascending aorta; after implantation, the pump is situated just above the aortic valve. The long cannula (26.2 cm) is designed for insertion through the femoral artery and fashioned so that the pump will be positioned in the descending thoracic aorta beyond the aortic isthmus after implantation. With this design, the cable is not curved when inserted distally from an arterial cutdown (Fig 1). Proper positioning of the Hemopump is confirmed by a chest roentgenogram, as required in the experimental protocol, or by fluoroscopic guidance.

Femoral Artery Approach

The most common method of Hemopump implantation is through the left femoral artery approach. The cannula and pump may be introduced directly into the arteriotomy with the 12-mm woven Dacron graft over the drive cable; the graft is then sutured to the femoral artery (Fig 2A). This technique is particularly useful when the artery is of marginal size (7- to 8-mm diameter).

The usual approach (Fig 2B) is to anastomose the graft to the artery at the site of the incision. The graft must be of sufficient length to allow introduction of the entire

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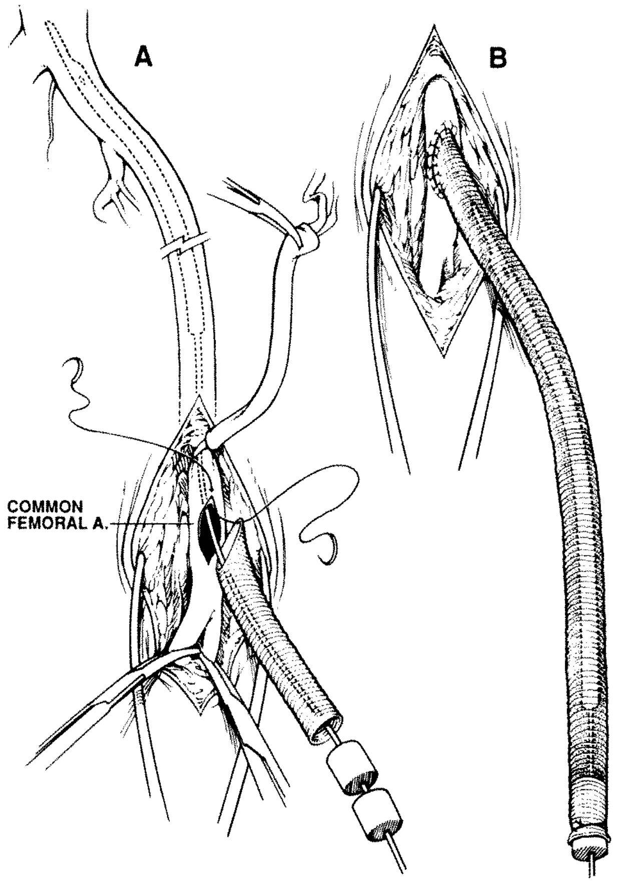

Fig 2. Two different techniques for femoral insertion. (A) Direct advancement of the pump through the arteriotomy, followed by graft anastomosis. (B) Advancement of the pump through anastomosed graft.

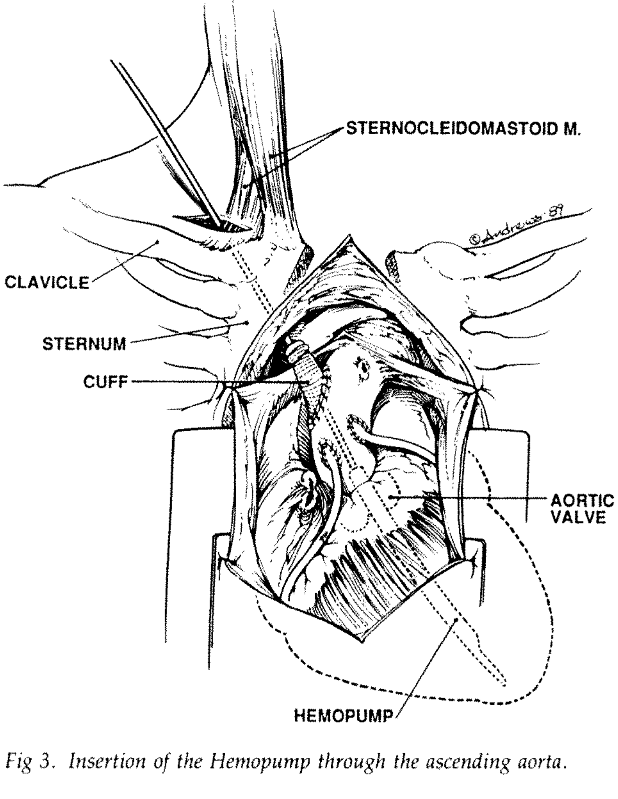

Fig 3. Insertion of the Hemopump through the ascending aorta.

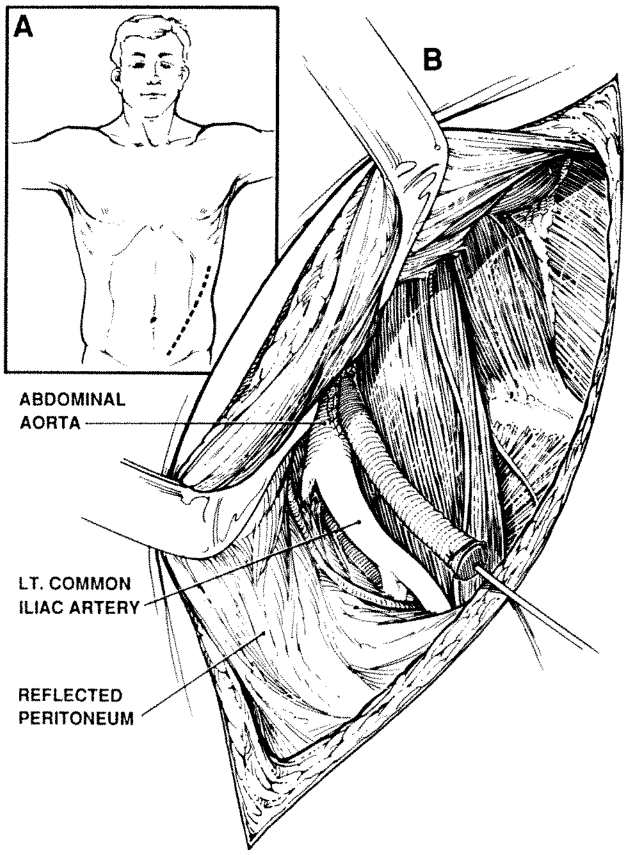

Fig 4. Retroperitoneal approach to the distal abdominal aorta and iliac vessels.

pump and cannula assembly. A silicone plug, which is positioned at the end of the graft, allows the cannula to be manipulated without causing bleeding. The Hemopump is then introduced into the aorta and, by use of fluoroscopic guidance, the cannula is positioned in the left ventricle. The graft is then trimmed to allow closure of the skin over the Dacron. Fluoroscopic guidance can be avoided in patients who have undergone sternotomy and confirmation of device placement can be accomplished by palpation of the aorta and left ventricle.

Failure of insertion occurred in 4 patients who had diffuse arteriosclerotic changes. No vascular complications resulted from the insertion attempts in this group of patients.

Thoracic Approach

Thoracic insertion of the Hemopump is useful in patients who cannot be weaned from cardiopulmonary bypass by conventional means and who meet the criteria for receiving additional support. The ascending aortic approach can also be used in patients with severe peripheral vascular disease that precludes retrograde insertion of the pump through the femoral or iliac arteries or distal abdominal aorta.

Although implanting the Hemopump through the ascending aorta without use of cardiopulmonary bypass appears to be technically feasible, bypass was required for all patients in whom this approach was used because of other indications before pump placement. A side-biting clamp is placed on the anterolateral aspect of the distal ascending aorta, and a longitudinal aortotomy is made. A 12-mm woven Dacron graft, beveled to 60 degrees, is then sutured at the site of the arteriotomy. With the anastomosis complete, the open end of the graft should be facing cephalad. A small, separate stab incision is then made in the right supraclavicular region. The Hemopump is inserted through the stab incision, passed under the sternum, and introduced into the graft. A specially designed silicone plug is placed around the drive cable and advanced into the graft. A heavy ligature is secured around the graft and plug to prevent bleeding (Fig 3). Brief discontinuation of cardiopulmonary bypass and manual compression of the ventricle may be necessary to facilitate opening of the aortic valve and allow positioning of the Hemopump in the left ventricle. The position of the cannula in the left ventricle and aorta can be confirmed through palpation, but fluoroscopic guidance is useful.

Currently the thoracic approach is not being used because cable fractures occurred during the weaning process in 3 of the early patients. All patients survived, and there were no related complications. A new cable has been developed for thoracic placement and should be ready for use in the near future.

Retroperitoneal Approach

In patients whose femoral artery is too small for insertion of the Hemopump, implantation can be accomplished through the common iliac artery or distal abdominal aorta. The surgical approach is through a standard flank incision (Fig 4). The pump is then inserted through a Dacron graft, as described.

Conclusion

The Hemopump has proved successful in providing left ventricular support in patients with cardiogenic shock from diverse causes [2]. Because of the pump's unique design and its ability to be inserted through the femoral artery, ascending aorta, iliac artery, or abdominal aorta, it potentially can be used in many clinical situations in which left ventricular support is required.

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Lord Brock—The Direct Approach

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The contributions of Sir Russell Brock to the early development of cardiac surgery are multiple. However, one of the most telling was his description of 3 cases of pulmonary stenosis in 1948 treated by direct transventricular valvulotomy. His subsequent treatise, *The Anatomy of Congenital Pulmonary Stenosis*, published in 1957, outlined this direct approach to cardiac lesions, a philosophy commonly adopted in modern care of cardiac lesions and a major contribution to cardiac surgical thinking.

(Ann Thorac Surg 1989;48:736–7)

In 1957, Sir Russell Brock published his classic treatise *The Anatomy of Congenital Pulmonary Stenosis* [1]. In the introduction, he noted that "the greater part" had been written more than 5 years earlier; however, its publication "was deliberately delayed" to gain a "fuller experience . . . of greater value," and he had "not regretted" the delay. Lord Brock reasoned as follows:

> [I]t is more satisfactory to know why a thing occurs, and it seemed obvious that there must have been an explanation for the common occurrence of such a peculiarly constant form of maldevelopment as pulmonary stenosis, even though its varieties are several.
>
> Second, his interest in the relief of pulmonary stenosis by *direct operation* made it essential to understand the anatomic malformation, both of the stenosis itself and of the muscular structure of the ventricular walls.

Surgical experience has confirmed these observations both in this lesion and in the concept of the direct approach [2].

Brock further recognized that the right ventricle is not a "rather simple muscular structure which expands to receive blood from the atrium and then contracts to expel it."

> In fact, the arrangement is much more complex than this. The formation, structure and function of the outflow portion of the right ventricle in particular is quite complex and is of great practical importance in relation to the stenoses that affect it.

This treatise and the articles that preceded it, in effect, proposed the current concept of a logical surgical approach to direct operative relief of pulmonary obstructive lesions [1–3].

Obstructive lesions of the right ventricular outflow tract represent both the simplest and most complex congenital malformations the cardiac surgeon must treat. The spectrum of disease from mild valvar stenosis to total atresia may be characterized by the term "obstruction." Yet because of this spectrum, a multiplicity of procedures (such as Blalock-Taussig, Potts, Waterston, Cooley) were developed by the surgeons of Brock's era, each designed to alter the physiology and allow survival [5–8]. Rather than addressing the embryologic defect by a direct operative approach to the obstruction, they addressed the life-threatening symptom of "cyanosis," in many cases creating permanent alterations in both anatomy (pulmonary artery distortion and obstruction) and physiology (pulmonary arterial hypoplasia or pulmonary vascular disease), obviating the possibility of repair [9]. Clinics became filled with shunted patients relieved of their cyanosis but destined to a slow inexorable fate, with only a few excellent candidates for repair. Refinements in alternate methods cannot be denied; however, the philosophy of direct operation remains both appealing and justified by the results of recent years (including that of balloon valvoplasty). Early repair of right ventricular outflow tract obstruction has become the standard when possible, whereas central palliation provides relief of the physiological defect, cyanosis [10] (Fig 1). Furthermore, as noted by Brock, if this is not performed, "no relief is given to the burdened right ventricle and an additional strain is thrown on the left ventricle in the form of shunted blood, such that severe hypertrophy and endocardial ischemia may be manifest."

Brock claimed that direct operations on the stenosis were better performed at an early age so that the pulmonary outflow tract could grow with the patient.

> Development must be progressively, severely arrested if the stenosis is not relieved, for, in addition to the stenosis becoming relatively more severe as the patient grows older, it becomes absolutely more severe as a result of the daily wear and tear of the stenosis passage. Ultimately, the obstruction may become complete, or so nearly complete as to be virtually so. . . . Valvulotomy allows progressive dilatation of the main pulmonary artery and of its branches. Infundibular resection, by correcting the tethering of the walls of the infundibulum by the fibrous ring which loops around the crista supraventricularis, allowing growth and expansion of the channel as the rest of the heart grows. In other words, normal growth and progressive improvement are favored.

Brock's clinical experience demonstrated that even a severely underdeveloped pulmonary outflow tract could be restored to full growth by a successful direct operation. Although the techniques were of his era (closed valvulotomy using a valvulotome and blind muscular resection) he believed:

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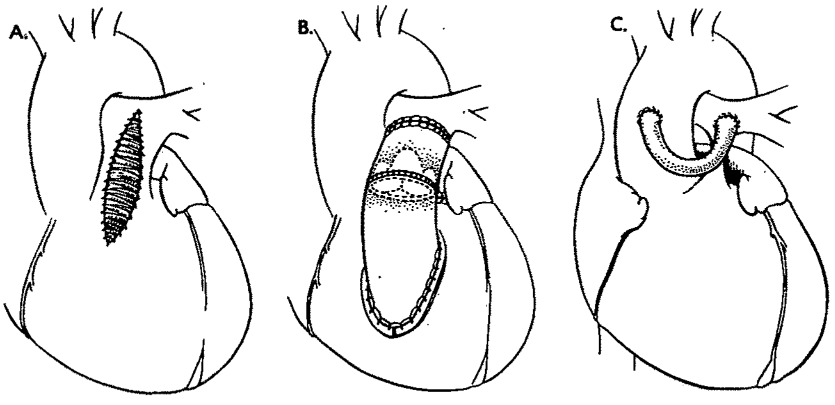

Fig 1. Current methods of central palliation. (Reprinted from [2] by permission of The C.V. Mosby Company.)

> The day must surely come when direct operations upon the substance and structure of the heart will be as firmly established and as successfully conducted as operations upon the lungs, brain, and other major systems en vitro that for so long defeated our efforts. There can be little doubt that there will be fundamental changes in technique such as possibly those associated with temporary discontinuation of the circulation through the heart while it is being operated upon. In the meantime, we must attack the problem and begin the development of a technique for intracardiac operations, for unless begun, the task can never proceed.

This statement prefaced Brock's description in the *British Medical Journal* in June 1948 of 3 successful cases of closed pulmonary valvulotomy using the transventricular approach and a curved valvulotome [3]. Current surgical experience confirms Brock's experience, in both simple obstructive lesions and more complex problems requiring central palliation or muscular resection [2]. Of note in his treatise, the concept of resection of the infundibular muscle and the crista is even proposed, predating current methods of ventricular septal resection in more complex outflow tract lesions as well as enlargement of the bulboventricular foramen in single ventricle lesions.

The concepts of Brock remain sound, attesting to the deliberate and methodical approach he espoused in his pursuit of the direct approach. In the introduction to *The Anatomy of Congenital Pulmonary Stenosis*, Brock gave credit to his predecessors, his work finding its inspiration in the lectures by Sir Arthur Keith published in *The Lancet* from 1904 to 1924. At the Brompton Hospital, his approach of close attention to the anatomy and detailed embryologic development of this lesion paralleled his approach to his life's work [11]. His eulogy, published in the *Annals of the Royal College of Surgeons of England* in 1980 [12], described him as "the master" as the result of the "research," "clear presentation," and "accuracy of his findings . . . the qualities of which are revealed in the anatomy of pulmonary stenosis." For surgeons today, these qualities remain as an example, and the direct approach is much appreciated.

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Transvenous Pacing: A Seminal Transition From the Research Laboratory

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In the late 1950s, nonsurgical catheter therapies for the treatment of various disorders did not exist, although surgery was at its highest level of development in the classic sense: extirpation of organs, incision and drainage, diagnostic biopsy. Today we live in an era of diagnostic and therapeutic methods based on catheters: observation, aspiration, drainage, stretching, and manipulation by means of relatively atraumatic tubes. The development of catheter techniques has spawned undreamed-of specialties, industries, and professional societies. Although catheters had been used before then in research laboratories, Furman's clinical application of electrode-tipped venous catheters in treating patients with complete heart block and Stokes-Adams seizures represents a turning point. This advance may be regarded as the catalytic and seminal event for the growth of catheter technologies, which are so prominent in the world of medicine today, and their application to many fields of medicine and surgery.

(Ann Thorac Surg 1989;48:738–40)

A turning point in history may be instantly and explosively evident, such as the launching of the Soviet Sputnik in 1957, but more often than not it is identified only in retrospect—an arbitrary decision, a matter of opinion. We propose that it was just such a moment when Furman and Robinson [1] first used a transvenous catheter-electrode to treat a patient with complete heart block and Morgagni–Adams–Stokes syndrome. This is not to ignore Zoll's [2] wonderful achievement 5 years earlier, when he demonstrated that stimulation of the heart through the intact chest could be lifesaving, for this in itself, perhaps in combination with closed chest cardiopulmonary massage, inaugurated effective worldwide cardiac resuscitation programs. However, it seems to us that the therapeutic use of a cardiac pacing catheter introduced an entirely different and new aspect of cardiology, which in many ways spawned new specialties and industries. Certainly, it can be identified, along with Zoll's earlier work, as the beginning of the era of cardiac pacing. Furman surely will regard this thesis as ridiculous, but even before being asked to comment on his early contribution, this idea had occurred to us.

Previously, catheter electrodes had been used almost exclusively in university research laboratories, without a vision of future therapeutic possibilities. His-bundle electrograms had not yet been discovered, and only the gross details of conduction pathways through and around the atrioventricular node had been described. Pathways of rapid and slow atrioventricular conduction and aberrant pathways, although deduced to exist by electrocardiography geniuses of the past, were yet to be specifically identified and localized electrically. Although the idea of indirect stimulation of the heart had been known almost since the time of Galvani (readers may be fascinated by the counterpart of Alexis St. Martin, a certain Catharina Serafin, whose partially exposed heart was stimulated directly by Von Ziemssen in 1882), and although a prototype external pacemaker had been developed by Hyman in 1934, and although others had achieved stimulation of the heart, no one had yet demonstrated that cardiac stimulation was possible as a routine lifesaving measure [3, 4]. In short, this event represented the critical transition from research to clinical application.*

The 30 ensuing years saw a cascade of discoveries and therapies based on the use of an intracardiac catheter of one sort or another to stimulate, sense, explore, puncture, obstruct, destroy, and even to visualize cardiac structures (Fig 1). The world of cardiology became catheter-oriented, propelled by parallel innovations that simplified the use of such devices. Notable were the Seldinger technique for transcutaneous vascular access, introducers, and self-sealing sheaths that permit the bloodless exchange of probes and many other innovative devices. The methodology spread across specialties, stimulating the development of invasive cardiology and radiology. One might even make a case for its influence on gastroenterology,

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* Furman's use of a transvenous electrode for cardiac stimulation was not the first time this had been conceived of as a treatment modality. It had been used in animals for many years, notably in efforts to maintain atrioventricular synchrony [5, 6]. Kossmann, in fact, never got around to reporting his experience in the mid-1950s of treating what probably really was the first heart block patient with an intravenous pacing electrode (Kossman CE, personal communication, June 1989). He used a transvenous bipolar pacing wire that had been developed for him by the US Catheter Corporation in the late 1940s, and described by him and his associates in 1950 [7]. The patient had been treated for several days with Zoll's external transthoracic pacemaker to the point of physical exhaustion from the continuous chest-wall contractions. Once pacing was taken over by the transvenous wire and the external device had been turned off, the patient fell asleep for the first time in days. The ultimate outcome of that patient's treatment is unknown, although in the absence of permanent pacing it undoubtedly was not good. (The records from that patient's hospitalization at Lenox Hill Hospital are being sought, but with little hope of finding such an old file.)

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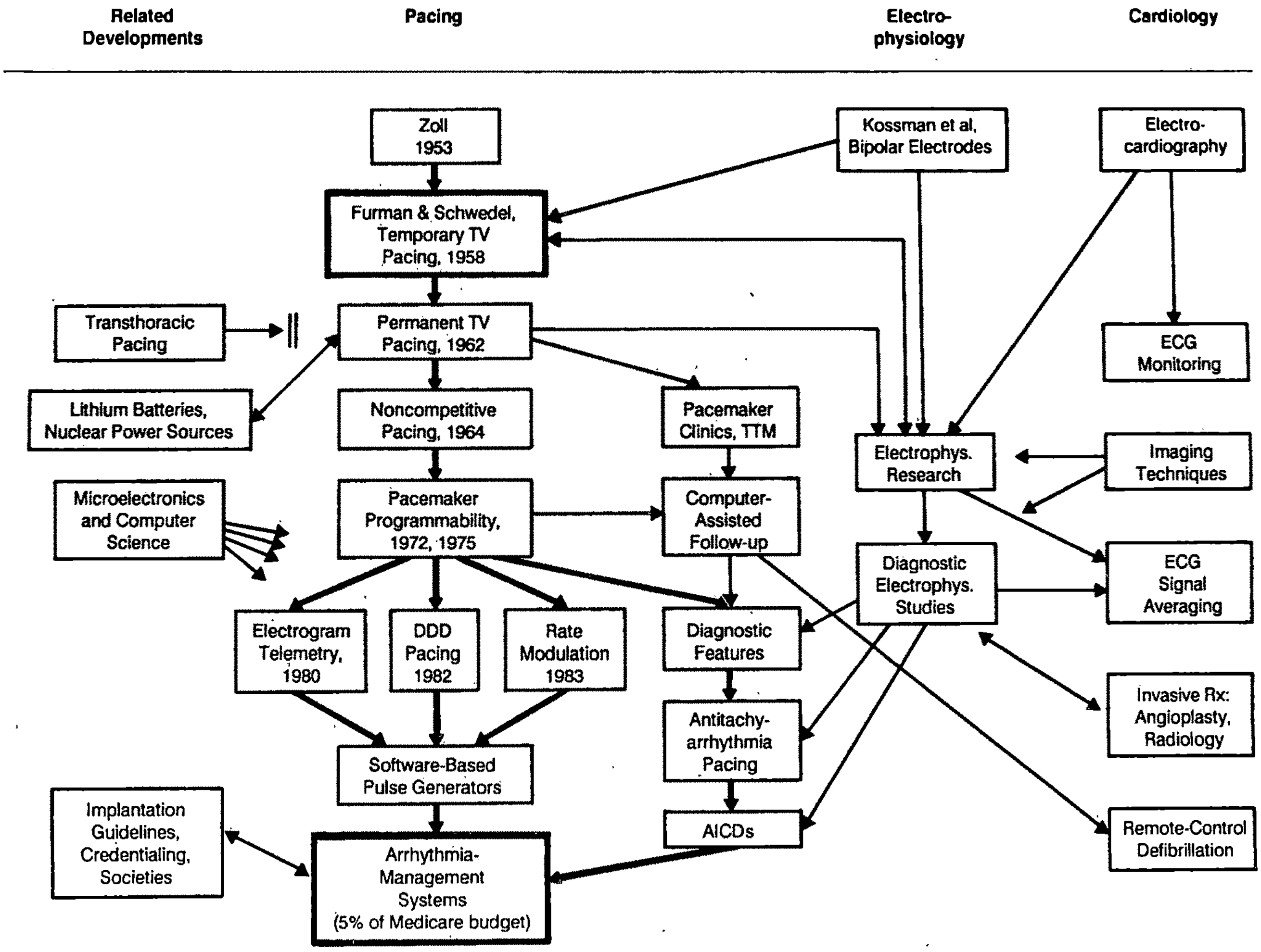
Related Developments
Pacing
Electro-physiology
Cardiology
Zoll 1953
Kossman et al, Bipolar Electrodes
Electro-cardiography
Furman & Schwedel, Temporary TV Pacing, 1958
Transthoracic Pacing
Permanent TV Pacing, 1962
ECG Monitoring
Lithium Batteries, Nuclear Power Sources
Noncompetitive Pacing, 1964
Pacemaker Clinics, TTM
Electrophys. Research
Imaging Techniques
Microelectronics and Computer Science
Pacemaker Programmability, 1972, 1975
Computer-Assisted Follow-up
Diagnostic Electrophys. Studies
ECG Signal Averaging
Electrogram Telemetry, 1980
DDD Pacing 1982
Rate Modulation 1983
Diagnostic Features
Invasive Rx: Angioplasty, Radiology
Antitachy-arrhythmia Pacing
Software-Based Pulse Generators
Implantation Guidelines, Credentialing, Societies
AICDs
Remote-Control Defibrillation
Arrhythmia-Management Systems (5% of Medicare budget)

Fig 1. Developments in pacing and related fields over a 30-year period. (AICDs = *automatic implantable cardioverter defibrillators;* DDD = *dual-chamber demand;* ECG = *electrocardiographic;* Rx = *prescription;* TTM = *transtelephonic monitoring;* TV = *transvenous.)*

urology, oncology, and general surgery, all of which utilize transcutaneous intervention.

The temporary pacing lead became a standard means of rescuing patients with complete heart block; in 1962 it was modified for use in permanent pacemaker implants [8, 9]. New pacing indications became evident in which treatment was directed not only toward extending life but also toward improving its quality. The simple application of the pacing catheter for an ever-widening variety of conditions permitted its use in hospitals of any size and by clinicians of many specialties, and fostered the implantation of permanent pacemakers in millions of patients throughout the world.

Electrodes such as those used in pacing became more sophisticated in the 1970s and 1980s, and even permitted intracardiac diagnosis. With the growth of electrophysiology, it became evident that electrical devices could also be used to identify, prevent, and even interrupt tachyarrhythmias. This led to the development of antitachyarrhythmia devices capable of treating arrhythmias by electrical stimulation alone. Eventually, a seemingly unattainable dream came true with the automatic implantable defibrillator, a device capable of shocking the fibrillating heart back to a normal rhythm. Catheters are now used for the entirely new field of endocardial ablation of atrioventricular conduction pathways.

The techniques of electrophysiology soon were incorporated in pacemakers themselves. With the parallel development of long-lived batteries and software-based pulse generators, it was possible to use the implanted pacemaker itself for diagnostic purposes. Pacemaker pulse generators store an enormous variety of information and on demand transmit it to the outside world via noninvasive electronic telemetry. Poised for clinical trial, devices exist that combine all of the aforementioned technologies so that it will be possible to pace the heart for a bradyarrhythmia, to interrupt an incidental tachyarrhythmia with low-energy stimuli or defibrillating shocks, and to transmit a high-resolution intracardiac electrogram from the atrium, the ventricle, or both. Thus, the pacemaker has become an implanted arrhythmia-management system.

Pacemakerlike devices connected to intravascular catheters, using programmability techniques originally developed for pacing, have made it possible to instill drugs automatically into the bloodstream, celomic cavities, and the central nervous system for the treatment of tumors, diabetes, and intractable pain. Electrical stimulators are used to enhance bone healing and to reduce postoperative pain. Minute catheters have been introduced into every body space, even the anterior chamber of the eye.

Once it became recognized that electrical probes could be used to identify intracardiac electrical events, attempts were made to monitor such events transcutaneously. It was Furman himself, in fact, who, with his associates, first described the transtelephonic monitoring of pacemakers [10], an element in a growing industry of remote cardiac monitoring. Monitoring has since been extended to distant radio receivers so that, from a central command station, intensive care unit technicians can observe dozens of patients simultaneously. Remote monitoring via radio and conventional or digital cellular telephones now makes it possible to defibrillate patients miles away from the treating physician.

With the development of pacing and its spin-offs, the need for new subspecialty societies became evident, notably in cardiac pacing and electrophysiology. Already there are plans for subspecialty board examinations in electrophysiology, and there is an existing examination for competence in pacing, organized through the North American Society of Pacing and Electrophysiology [11]. That society, now in its 10th year, was a direct outgrowth of the advances in cardiac stimulation and sensing.

The socioeconomic impact of cardiac pacing has been immense. If the implantable defibrillator becomes as widely used as seems likely, it is possible that there will be as many defibrillators implanted in this country as there are pacemakers. The cost to the public for patient selection, in-hospital workup, diagnostic procedures, drug testing, and the devices themselves soon will reach $6.9 billion a year, and consume 5% of the Medicare/Medicaid budget. Imagine the percentage of the budget that will be accounted for by *all* invasive procedures, such as balloon angioplasty, atherectomy, and valvoplasty!

Furman is not responsible for all of this, although we blame him for some of it. Certainly he is not aware of our hypotheses. But this is the stuff of which milestones are made. When reviewing the impact of his work, one sees the entire panoply of cardiac pacing and its ramifications, and recognizes the debt owed him by the medical community.

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Lung Transplantation

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CORRESPONDENCE

Bovine Pericardium: Another Cautionary Note

To the Editor:

Bovine pericardium has been shown to be a useful prosthetic material in the repair of simple and complex congenital heart defects. Commendable properties include its availability, flexibility, strength, and ease in handling and sewing. However, vigilance in follow-up was advised as to possible calcification and shrinkage with changes in size and shape [1]. Subsequently, pulmonary venous obstruction was reported with its use in the Mustard procedure [2].

Since October 1986, 5 patients (aged 3 weeks to 6 months) with simple complete transposition of the great arteries underwent the Senning procedure with bovine pericardium augmentation of the pulmonary venous channel. Within 4 to 9 months, all showed evidence of pulmonary venous obstruction. Symptoms included tiring with feeding, poor weight gain, irritability, diaphoresis, and cough. Clinically, low-pitched long systolic to continuous murmurs were noted over the right side of the chest. Pulmonary venous congestion was present on chest roentgenograms. Two-dimensional echocardiography with pulsed Doppler showed narrowing at the isthmus of the neo-left atrium with a turbulent high-velocity systolic jet. Cardiac catheterization demonstrated clinically significant pressure gradients between the posterior and anterior segments of the neo-left atrium or between simultaneous pulmonary artery wedge and right ventricular end-diastolic pressures. Midbaffle obstruction was noted on cineangiography. Reoperation with baffle revision was necessary in all patients.

Histological examination of the excised bovine pericardium with exuberant peel formation revealed fibrous tissue with a myxomatous stroma, granulation tissue, chronic inflammation, foreign body giant cells, and calcification.

Those who continue to use the atrial switch procedure for correction of complete transposition of the great arteries must be cognizant of the association of pulmonary venous obstruction with the use of bovine pericardium.

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Pericardial Substitutes

To the Editor:

I thank Dr Heydorn and associates for their article on pericardial substitutes [1], in which they were able to collect the opinion of a large number of cardiac surgeons on this subject. I was very impressed by the conclusive comment that "none of the materials listed was reported to be completely successful in facilitating reoperation."

I agree with this conclusion, and for this reason I have elaborated an original technique of pericardial closure with a pleural flap. The technique [2] is different from that mentioned in their report [3] and used by thoracic surgeons, because the mediastinal pleura is used instead of the parietal pleura.

More than 20 patients have been operated on with this procedure, but none of them has been reoperated on so far. The principle, however, has already been demonstrated to give good results because a vital autologous flap is used to close the pericardium.

The most relevant thing is that the mesothelial surface of the pleural flap remains in contact with the epicardium, and therefore two main advantages can be achieved in case of resternotomy: (1) the heart and the coronary grafts are protected during the reopening of the sternum even in emergency operations, and (2) a safe plane of dissection can easily be found, facilitating the identification of the intrapericardial structures, including grafts and native coronary arteries. For these reasons I believe I will continue to use this method instead of using other kinds of pericardial substitutes.

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Reply

To the Editor:

We appreciate the comments of Dr Massimo. He refers to the article by Shechter and associates that describes the use of parietal pleura to close defects in the pericardium resulting from intrapericardial ligation and division of the pulmonary vasculature during pneumonectomy. Its purpose is to prevent cardiac herniation.

The concept of using autologous pleura to construct a new pericardial sac to prevent adhesions to either epicardium or sternum is a valid one. Evaluation of the efficiency of Dr Massimo's technique will be dependent on close observation and a description of the findings should reoperation become necessary.

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Pneumothorax in Cystic Fibrosis

To the Editor:

Spector and Stern [1] have reviewed the experience accumulated over 30 years at their institution with the treatment of pneumothorax in cystic fibrosis patients and have made recommendations that seem to conflict with their own data.

The patients treated by pleurectomy had a 5% primary failure rate and 11% recurrence rate. Two of these patients (3%) died during the hospitalization for treatment of pneumothorax, although 1 of these died of a pneumothorax on the contralateral side. The total major morbidity and mortality of pleurectomy

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was, however, 19%. The ipsilateral recurrence rate with this treatment was 11% (6 of 57 patients), compared with the recurrence rate of 14% (4 of 28) when no treatment was used in their series. When comparing these results by χ^2 analysis there is no significant difference in recurrence rate between observation alone and pleurectomy ($p = 0.613$). Therefore, Spector and Stern's conclusion that pleurectomy is the treatment of choice for these patients does not seem to be substantiated by their data.

The patients in this review who were treated with talc (although it was administered in a suboptimal manner and apparently reserved only for the most debilitated patients) had 100% success. There were no documented recurrences, and no morbidity from the procedure, although 1 patient died ten days after talc administration, presumably of the underlying disease. Thus, it seems that their experience with talc instillation has been favorable, although limited. Therefore, the recommendation that this technique be reserved for "patients in severe respiratory failure who are unable to undergo an operation" is not supported by this report or by the published experience of others [2–7].

In summary, we feel that Spector and Stern's data support our conclusion that talc poudrage is a benign and very effective treatment for pneumothorax in patients with cystic fibrosis. We feel that administration of talc by thoracoscopy allows a uniform dispersal throughout the pleural space and virtually eliminates the possibility of recurrence.

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Reply

To the Editor:

We appreciate the opportunity to respond to the correspondence from Drs Rodgers and Tribble. Surgical partial pleurectomy had a 95% success rate for resolving the pneumothorax of 57 patients; three pneumothoraces failed to resolve and 2 patients died during their hospitalization, resulting in a combined morbidity/mortality of 9%. Surgical therapy for resolution of the pneumothorax was statistically ($p < 0.01$) better than observation, thoracostomy alone, or chemical pleurodesis using quinacrine, tetracycline, and $AgNO_2$, thus substantiating the fact that surgical partial pleurectomy is the treatment of choice compared with the above modalities.

Our experience with talc pleurodesis was limited to only 5 patients. It had a 100% success rate but 1 patient died during hospitalization, resulting in a combined morbidity/mortality of 20%.

Of the 5 patients in the article by Tribble and associates (their reference [7]), 40% needed general anesthesia. In our own experience with thoracoscopy, we found it technically difficult to visualize all areas of the lung in patients with adhesions and very stiff lungs who were under general anesthesia. We still feel that if a patient is to undergo general anesthesia, a minithoracotomy with oversewing of blebs and pleurectomy is better than talc pleurodesis alone. More experience is needed with talc pleurodesis before we can adequately determine the best therapeutic modality.

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Heart-Lung Transplantation for Pneumothorax in Cystic Fibrosis

To the Editor:

The article by Drs Spector and Stern [1] in a recent issue of *The Annals* concerning the management of pneumothorax in cystic fibrosis concluded that the treatment of choice was partial pleurectomy. At our institute heart-lung transplantation was first successfully performed for this condition in 1985 [2], and early results have been encouraging [3]. Fourteen patients with cystic fibrosis have received heart-lung transplants and 12 are alive at 3 to 42 months after operation. When our heart-lung transplantation program began in 1984, previous thoracic surgery was a contraindication to this procedure. Since then, however, in a series of 61 heart-lung transplant recipients, 5 have had previous thoracotomy and 1, median sternotomy without an increased incidence of postoperative chest wall bleeding. We have also successfully performed transplantation in patients with cystic fibrosis who are on steroid therapy or have insulin-dependent diabetes mellitus, features that were previously contraindications.

We have assessed 51 patients with cystic fibrosis from 26 centers in the British Isles for heart-lung transplantation. We are not assessing patients who have had a pleurectomy or pleurodesis because the risk of postoperative bleeding is considered unacceptably high. Because of this many of the centers who refer patients with cystic fibrosis are now managing recurrent pneumothoraces with minimal intervention [4].

Although in some patients surgical treatment of pneumothorax may be necessary for survival, we would advocate caution in treating all patients with cystic fibrosis who have pneumothorax in this way.

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Reply

To the Editor:

My colleagues and I congratulate Dr Smyth and associates on their excellent results with heart-lung transplantation in patients with cystic fibrosis. As heart-lung transplantation becomes more available, we may need to change our therapy in patients with cystic fibrosis. Our data suggest, however, that only 30% to 40% of patients with cystic fibrosis who have pneumothoraces will respond to minimal therapy. If 9% of the cystic fibrosis population will experience a pneumothorax, then only 3% of this group would benefit from minimal therapy and remain candidates for heart-lung transplantation. At the present time, with only a very small percentage of patients with cystic fibrosis actually undergoing heart-lung transplantation, this leaves us with an even smaller subset of patients meeting both criteria. We agree that all options including heart-lung transplantation should be discussed with all patients, but should we subject 60% to 70% of patients with cystic fibrosis who have a pneumothorax to prolonged hospital stays and higher complication rates secondary to multiple therapeutic courses to allow those very few patients heart-lung transplantation?

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Human Immunodeficiency Virus

To the Editor:

The articles by Frater [1] and Condit and Frater [2] about the human immunodeficiency virus (HIV) touch on a very important and sensitive point: disclosing the HIV carriers by voluntary testing. Should only voluntary testing be permitted? Perhaps. But, I believe, a patient who refuses it should face an ultimatum: no operation without testing. I wish to make an unorthodox suggestion: obligatory testing for patients who need an operation. The problem of obligatory preoperative testing for HIV can be reduced to a basic question. What is more important, the rights of an individual or the rights of the society? In North America the balance between the two is very strongly biased in favor of the individual. However, in reflecting on the problem of prevention of acquired immunodeficiency syndrome (AIDS), a question comes to mind. Should individuals be permitted to spread AIDS, often with total disregard for the rights of others? Many patients with AIDS are, in fact, doing just that. They continue to spread the deadly disease, knowing full well what they are doing, and disregarding the rights of others. There is almost no doubt that some patients without any sense of morality are doing it with an intentional *Schadenfreude*: "If I am going to die, let others go with me; I have the right to do what I am doing." Do they have the right to do it? Are the rights of an individual to privacy everything, and the rights of the society to protection nothing?

With all respect to the American tradition of individual rights, the concept must be established that the rights of an individual cannot be more important than the rights of thousands and millions of individuals. If, and when, preoperative testing for HIV becomes compulsory, appropriate precautions can and should be taken to assure that the information thus obtained will not be thoughtlessly or intentionally distributed where it should not be. As Dr Frater stated in his editorial, "It is in fact not difficult to devise procedures whereby, when the patient so desires, absolute confidentiality can be preserved, and information only revealed to appropriate other parties: (1) if a contaminating incident occurs, (2) if the information could be useful in understanding and managing the subsequent clinical course of the patient, or (3) if the information could be valuable in retrospective analysis of a group of patients." But above all, compulsory preoperative testing must be made lawful, both for protection of those who take care of the HIV carriers and for the protection of society at large. Health care workers also have their individual rights, and they are entitled to know whether they are at risk of acquiring the deadly virus while performing their duties. This is a matter of public health, perhaps not less important an issue than the rights of privacy.

The US is the world's leader in medicine today. The strict adherence to protection of privacy and rights of individuals indicates that this leadership applies also to the matter of ethics. It is, therefore, the US that must show the others how to prevent the spread of AIDS, and not only by technical and scientific means, but by appropriate legislation. The Society of Thoracic Surgeons should lead in the struggle for this legislation.

I realize that my point of view may sound somewhat unorthodox to those who learned to live with the principle of "privacy above all else," but differences of opinion have always been the main stimulating factor of progress in medicine. Perhaps for this reason, unorthodox points of view should be published and discussed.

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Reply

To the Editor:

I have read Dr Weissberg's letter with interest. He has focused on the critical issue of individual versus societal rights. New developments in thinking on AIDS have resulted in the institution of prophylactic treatment with 3'-azido-3'-deoxythymidine (AZT) for some carriers of the HIV. This produces an additional very important reason for easing the ability to test: it may be to the patient's advantage.

Recently I operated on an elderly gentleman in whom the routine serologic test for syphilis, still being performed, came back positive. This had to be reported to the City Health Department according to the system in operation for 60 years or more. I duly received a thick sheaf of further instructions so that the implications for this positive result to the patient and his contacts could receive appropriate attention for the proper maintenance of public health! I wish that we were as concerned about the HIV as we are about the spirochete.

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Blood Transfusion and Lung Cancer Recurrence

To the Editor:

There are currently five published reports that attempt to describe the effects of perioperative blood transfusions on recurrence of completely resected non-small cell lung cancer [1–5]. All are retrospective. Two series [3, 4] containing almost 600 stage I patients clearly demonstrated no relation between lung cancer recurrence and perioperative transfusion. The first publication to ascribe an adverse effect to perioperative blood transfusion involved 165 patients with stage I (T1/N0) disease [1]. A statistically significant difference between the transfused and untransfused groups was achieved only when 15 patients who had undergone pneumonectomy were included. Separate analysis of the 150 patients treated by lobectomy failed to demonstrate an adverse effect of perioperative transfusion. Hyman and associates [2] studied 105 patients with stage I and II non-small cell lung cancers and claimed to show decreased survival in transfused patients. However, stage (the most well documented and universally accepted prognostic factor) did not correlate with survival. Surely, the results of such a study must be interpreted carefully.

The recent paper by Moores and co-workers [5] further confuses the issue of lung cancer recurrence after perioperative transfusion. The lack of complete records, the inclusion of second primary tumors as lung cancer recurrences, and the failure to discuss the discrepancy between results calculated for the entire group unstratified and when stratified by TN criteria and histology combine to make the study results difficult to interpret. Of the 433 patients included in the control arms of the three Lung Cancer Study Group studies, 103 (24%) are not included in the analysis because their transfusion records were unavailable. Certainly there is potential for such a large group to affect significantly the results of this study. Second, primary lung tumors (defined as histologically distinct from the primary tumor) were included as recurrences. The logic behind this is unclear. Second primary tumors seemingly represent a separate and unrelated issue and should be analyzed independently from the recurrence of lung cancer. Finally, calculation of transfusion effect by grouping all stages together is as uninformative as reporting unstaged results of surgical and chemotherapeutic trials.

The true effect of perioperative blood transfusion on non-small cell lung cancer recurrence is unclear. Broad generalizations are misleading and do not help to clarify the issue.

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Reply

To the Editor:

Drs Keller, Groshen, and Kaiser raise four points: (1) the relation between lung cancer recurrence and postoperative transfusion is already known based on previous work, (2) missing data, (3) the inclusion of second primary tumors in our analysis, and (4) the proper method of adjusting for prognostic factors. We will reply to each of these in turn.

Keller and associates claim to cite studies that "clearly demonstrated no relation between lung cancer recurrence and perioperative transfusion." This is an overinterpretation of the available data. First, there is a considerable body of evidence cited both by Keller and associates and by us demonstrating that there is a biological effect of blood transfusion in cancer patients. The issue is not if this effect exists but rather, what is its clinical and statistical significance. Keller and colleagues report their own data as demonstrating a relative risk of recurrence of 1.24 in stage I patients and 1.92 in stage II patients (Table 2 in Keller et al, Cancer 1988;62:606–10). The effect of blood transfusion observed in their own patients is consistent in direction and magnitude with the effect that we report. Their failure to find statistical significance in their data is due to the modest size of the effect and their relatively low number of events resulting in poor statistical power—not to absence of the effect. Second, the exclusion of patients who had undergone pneumonectomy from these analyses is unnecessary and may not be justified. The extent of operation is positively correlated with receiving blood products and, consequently, exclusion of patients with more extensive procedures may eliminate many of those who received blood products, further reducing statistical power. In other words, the study design decision to exclude a more heavily transfused subset of patients may have misled Keller and colleagues. We have employed what we view as a more proper method, which is to include all patients and adjust the analysis for the extent of resection. Third, it is virtually impossible to demonstrate that a hypothetical relationship does not exist. Doing so requires enormous amounts of data and strong justifications of the kind that are usually not available in a clinical setting. Consequently, the assertion by Keller and associates that a relation has already been disproved is a statement of bias rather than fact.

The fact that blood transfusion information could not be obtained for some Lung Cancer Study Group patients is not a major concern in this setting. Missing data, per se, is not a problem, provided the data are missing at random. If there is no association between the outcome of interest and the absence of the data, the inferences are unaffected. What is a plausible mechanism linking recurrence or death and missing data? We have looked for differences in major prognostic factors among those individuals with blood transfusion information compared with those without. No significant trends or associations could be found (except that stage II/III patients had fewer missing data), suggesting that the data were missing at random and resulting in unbiased estimates.

When adjusted for the effects of TN status, abnormal alkaline phosphatase, and extent of operation (as in our Table 5), the effect of blood transfusion on recurrence excluding second primary tumors demonstrates a hazard ratio of 1.49, which is statistically significant ($p = .025$). Therefore, the issue of second primary tumors is moot.

Keller and associates referred to a "discrepancy between results calculated for the entire group unstratified and when stratified by TN criteria and histology." Apparently, they are concerned that the unadjusted effect of blood transfusion is quantitatively different than the effect controlled for other prognostic factors. This is neither discrepant nor difficult to interpret. They display a lack of understanding of the statistical methods used to account for the effect of blood transfusion in the presence of other prognostic factors. Two standard methods were employed: the stratified log-rank statistic and multivariate proportional hazards regression. Both of these methods demonstrate a modest, but statistically significant, adverse effect due to blood transfusion when adjusted for tumor size, nodal status, abnormal alkaline phosphatase, age, histology, and extent of operation. Thus, we have estimated the effect of blood transfusion on recurrence and survival while simultaneously accounting for the effects of other prognostic factors. This is not "uninformative" as Keller and associates suggest but is the proper way of doing such analyses.

In summary, our report is qualitatively and quantitatively consistent with a body of additional evidence including the published work of Keller and colleagues. We have demonstrated that perioperative blood transfusion has an independent and statistically significant adverse effect on recurrence and survival in Lung Cancer Study Group patients even after accounting for other prognostic factors. Missing data and inclusion of second primary tumors are not issues. Our patients met clearly defined eligibility criteria and were carefully staged and followed, and the statistical methods of analysis were standard. The problem that "further confuses the issue" is not our report but the unsophisticated interpretation by Keller, Groshen, and Kaiser.

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Pharmacological Responses of Internal Mammary Artery Grafts

To the Editor:

I read with interest the recent article by Kitamura and associates [1]. Their finding that the internal mammary artery bypass graft is resistant to both ergonovine and nitroglycerin stimulation in contrast to the native coronary circulation has important clinical implications. This observation is particularly interesting in view of the fact that spontaneous internal mammary artery graft spasm after coronary artery bypass grafting occurs more often than has been reported [2, 3].

I wonder if Kitamura and associates also compared the internal mammary artery bypass grafts with saphenous vein bypass grafts to coronary arteries in these regards. Such a comparison would be very important. If saphenous vein bypass grafts behave more like the native coronary arteries in their responses to ergonovine or nitroglycerin, or both, then internal mammary artery will be the conduit of choice for bypassing not only coronary arteries with fixed obstructions but also coronary arteries that develop spasm in variant angina.

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3. Blanche C, Chaux A. Spasm in mammary artery grafts. Ann Thorac Surg 1988;45:586.

Reply

To the Editor:

I appreciate the opportunity to respond to Dr Cheng. In our study [1], the left anterior descending coronary artery was the vessel with demonstrable spasm superimposed on the fixed organic stenosis. Stenosis was always found in a fixed place during nitroglycerin infusion. The saphenous vein grafts (SVGs) were used for other vessels not showing marked spastic reaction during ergonovine infusion. For this reason, the pharmacological interaction between SVG and recipient coronary artery was not examined.

Although results of coronary artery bypass grafting in patients with spasm have been unfavorable, with a high incidence of recurrent angina [2] and high early occlusion rate of the SVG [3], no study has so far been made on pharmacological reactivities of SVG and the coronary artery in this particular group of patients. The internal mammary artery (IMA) graft, at least when used as an in situ graft, is viable with pharmacological responsiveness [1] and developmental abilities of caliber and length responding to flow demand and patient growth [4]. Based on the characteristics of IMA grafts, we thought that the IMA graft would be able to maintain its patency even when graft flow demand was reduced in the absence of spastic obstruction of the coronary artery in variant angina, and consequently we used the IMA graft in variant angina. Although there are very rare case reports of SVG spasm induced by ergonovine [5], the majority of freed SVGs would not dynamically respond to the flow situation of the run-off vessel or pharmacological intervention. This may possibly explain the high SVG failure in variant angina with dynamic obstruction.

The comment as to whether the IMA can serve as a conduit for bypassing the coronary artery that develops spasm, but has otherwise normal angiograms in the "variant of the variant" angina described by Cheng and associates [6], is very interesting. If coronary artery spasm always occurs in the same localized place, IMA may work well. However, unless the pressure gradient produced either by fixed obstruction or a localized spasm continues, the IMA graft will show the so-called string phenomenon. Because the intracoronary pressure gradient will not continue to exist in variant angina with normal arteriograms, the IMA graft with physiologically high resistance (thin and long) may be rejected as an unnecessary collateral, resulting in a very thin stringlike graft. We believe that good graft function of the IMA can be expected when a hemodynamically significant stenotic lesion with a pressure gradient is present.

The internal mammary artery develops a spasm when it is dissected and manipulated during operation; papaverine counteracts this spasm. Postoperative spontaneous IMA spasm may occur, as reported by Sarabu and associates [7], but without direct demonstration of this phenomenon either by angiography or direct vision during operation, it is very difficult to distinguish IMA spasm from recipient coronary artery spasm. There are not yet sufficient data to discuss spontaneous IMA spasm, but certainly considerable attention should be directed to it by surgeons. However, there is the question as to why early postoperative graft closure or spasm becomes detrimental enough to produce fatal myocardial ischemia in the absence of coronary artery spasm, considering that the patient survived the preoperative period without the graft. Original collateral flow or flow through the stenotic coronary artery still remains in the early postoperative period, particularly in patients with stable angina pectoris.

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Thoracotomy Through the Auscultatory Triangle

To the Editor:

Horowitz and associates [1] have recently rediscovered thoracotomy through the auscultatory triangle. My colleagues and I found this approach very useful and published our opinion approximately 30 years ago [2].

In our experience a long skin incision is not only unnecessary, but more painful than the abbreviated one we illustrated. Although the auscultatory triangle can be developed by blunt dissection, incision of a small portion of the latissimus, trapezius, and rhomboideus major causes no more disability than stretching them. It is almost never necessary to incise the serratus major. It is not necessary to remove a rib if the intercostal incision is made long enough. The latter can be done from the inside of the chest and causes surprisingly little discomfort.

I'm sure that the recent Editor's Note drawing attention to the "Ingelfinger Rule" [3] is pertinent in this case.

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EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information.

Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Twenty-fifth Anniversary—Open Heart Surgery, Jerusalem, Israel—December 28, 1989–January 1, 1990

For information on this meeting, contact Joseph B. Borman, MD, or the Secretariat, PO Box 32390, Jerusalem 91323, Israel; or telephone 972-2-663171 (telex: 26205 TRGBL IL; facsimile: 972-2-665277).

Specialty Review in Thoracic Surgery, Chicago, Illinois—January 8–13, 1990

This course is sponsored by the Cook County Graduate School of Medicine and is accredited for approximately 47 hours in category 1 of the Physician's Recognition Award of the AMA. For information, contact the Registrar's office: within Illinois, 1 (800) 621-4649; outside Illinois, 1 (800) 621-4651.

New Trends in Aortic Valve Surgery (Auto, Allo, Xenografts and Reconstruction), Riyadh, Saudi Arabia—January 17–18, 1990

For information on this workshop, contact Department of Cardiovascular Diseases, King Faisal Specialist Hospital & Research Centre, PO Box 3354, Riyadh 11211, Saudi Arabia (facsimile: 966-1-441-4839).

Esophageal Disorders: Pathophysiology and Treatment, Maui, Hawaii—January 25–30, 1990

This program is sponsored in part by educational grants from Synectics Medical, Inc, and Smith Kline & French Laboratories, and is accredited for 24 hours in category 1 of the Physicians Recognition Award of the AMA. For information on this meeting, contact Department of Surgery, Creighton University, CME Program, 601 North 30th St, Omaha, NE 68131; or telephone (402) 280-4501 (facsimile: (402) 280-4593).

Twenty-seventh Annual Seminar—Coronary Atherosclerosis: New Diagnostic and Therapeutic Approaches, Orlando, Florida—January 28–February 1, 1990.

This seminar is sponsored by the Departments of Radiology and Cardiology, Mount Sinai Medical Center of Greater Miami, Miami Beach, FL, and is accredited for 20.5 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Lucy R. Kelley, Program Coordinator, Radiology Seminars, Inc, PO Box 143762, Coral Gables, FL 33114-3762; or telephone (305) 674-2810.

Nineteenth Annual Meeting of the German Society for Thoracic, Cardiac, and Vascular Surgery, Bad Nauheim, Federal Republic of Germany—February 22–24, 1990

For information on this meeting, contact Hagen D. Schulte, MD, Professor of Surgery, Department of Thoracic and Cardiovascular Surgery, Heinrich-Heine-University, Moorenstrasse 5, D-4000 Dusseldorf, Federal Republic of Germany.

Twenty-ninth Annual Waring-Durrance Chest Conference, Aspen, Colorado—March 7–10, 1990

This meeting is sponsored by the Colorado Trudeau Society and the American Lung Association of Colorado. For information on this meeting, contact American Lung Association of Colorado, 1600 Race St, Denver, CO 80206.

Thirty-ninth Annual Meeting of the American College of Cardiology, New Orleans, Louisiana—March 18–22, 1990

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone 1 (800) 253-4636.

Aortic Surgery Symposium II, New York, New York—May 4–5, 1990

This symposium is sponsored by Meadox Medicals, Inc, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA and toward the New York Medical Association Certificate in Continuing Medical Education. For information, contact Beverly Farrier, Symposium Coordinator, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660.

Seventieth Annual Meeting of The American Association for Thoracic Surgery, Toronto, Ontario, Canada—May 7–9, 1990

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Third International Symposium on Echocardiography and Doppler in Cardiac Surgery, Vienna, Austria—May 9–12, 1990

For information on this meeting, contact G. Maurer, MD, or W. Mohl, MD, PhD, c/o Interconvention, A-1450 Vienna, Austria; or telephone +43/1/23 69/2642 (telex: 11 18 03; facsimile: +43/1/23 69/648).

Eleventh Annual Scientific Session of the North American Society of Pacing and Electrophysiology, San Diego, California—May 31–June 2, 1990

For information on this meeting, contact the North American Society of Pacing and Electrophysiology, 13 Eaton Court, Wellesley Hills, MA 02181; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

Eighth Annual Uniformed Services University of Health Sciences Cardiothoracic Surgery Symposium, Washington, DC—May 31–June 2, 1990

For information on this meeting, contact Fred H. Edwards, MD, Department of Thoracic Surgery, Walter Reed Army Medical Center, Washington, DC 20307-5001; or telephone (202) 576-1433.

The Society for Vascular Surgery/International Society for Cardiovascular Surgery, Los Angeles, California—June 4–6, 1990

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Sixteenth Annual Meeting of The Western Thoracic Surgical Association, Coronado, California—June 20–24, 1990

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

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SITUATIONS AVAILABLE

Cardiothoracic surgeon needed for rapidly expanding program located in a coastal area of the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Respond with curriculum vitae and names of references.

Please respond to Box 102. 102G/C

Cardiothoracic and vascular surgeon, board eligible/certified with experience in adult cardiac thoracic and vascular surgery, wanted to join well established 2-surgeon practice located in South Florida. Send CV.

Please respond to Box 105. 105C/B

Cardiothoracic surgeon to join established group in northwest Indiana (very near Chicago). Excellent salary. Please respond with curriculum vitae.

Please respond to Box 104. 104I/L

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join a large cardiac surgery group in southern California. Must have a minimum of 1 year experience in pediatric and neonate cardiac surgery (including fellowship). Excellent fringe benefits. Respond with curriculum vitae.

Please respond to Box 144. 144E/D

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 172. 172G/A

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/A

Cardiovascular and thoracic surgeon: Position available for a board certified or eligible cardiovascular and thoracic surgeon to join a 3-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association will lead to competitive salary, excellent fringe benefits, and early partnership. Please send curriculum vitae.

Please respond to Box 192. 192H/K

Cardiovascular and thoracic surgeon, BC/BE, wanted for established 3-man group in northeastern Ohio. Adult cardiac, vascular, and thoracic experience needed. Please send curriculum vitae.

Please respond to Box 195. 195I/K

Cardiovascular and thoracic surgeon, BE/BC, to join established private practice in San Francisco Bay area. Practice includes adult cardiac, thoracic, and vascular surgery. Initial contact should include curriculum vitae and references.

Please respond to Box 197. 197I/K

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another physician in private practice in Northeast. Immediate availability. Please respond with curriculum vitae.

Please respond to Box 198. 198I/K

Thoracic-vascular surgeon: Excellent opportunity for a board-certified or board-eligible noncardiac thoracic-vascular surgeon to join group practice on south shore of Long Island. Associated with two large level II 400-bed hospitals, fine recreational facilities, including beaches and boating, quality shools, and excellent homes.

Please respond to Box 148. 148EGIKA

Thoracic/general surgeon, BC/BE, to join board certified thoracic/generalist in a busy noncardiac thoracic and general practice. Present physician is the only thoracic surgeon in this community of 80,000 which has a service population of 250,000. Practice is ideally located just one hour from a major metropolitan area and only 30 minutes from an international airport. First year guarantee, incentive, malpractice, and health benefits provided.

Please respond to Box 201. 201J/L

The University of Florida is seeking a cardiovascular surgeon to join its major teaching unit in Jacksonville, Florida–University Medical Center. Candidates should be eligible for, or certified by, the American Board of Thoracic Surgery. We are looking for someone with excellent surgical skills and a strong interest in research and teaching. Clinical experience in pediatric cardiac surgery is highly desirable. This is a tenure-accruing position with academic appointment at the Assistant Professor to Associate Professor level depending upon qualifications. Applicant recruiting deadline is November 1, 1989. Anticipated starting date is November 15, 1989.

Applicants are invited to forward curriculum vitae and names and addresses of three references to Eric L. Ceithaml, MD, Chairman, Cardiovascular Search Committee, University Medical Center, 655 West 8th St, Jacksonville, FL 32209. Affirmative action/equal employment opportunity employer. 210JK

Thoracic surgeon, board certified/board eligible, wanted to join board certified thoracic surgeon in a 35-physician private practice multispecialty group in a waterfront community on Tampa Bay. Very active general surgery, vascular, and general thoracic practice.

Please respond to Box 214. 214J/L

Thoracic surgeon. A 220-physician multispecialty prepaid group in Hawaii is seeking a qualified MD to accept the second thoracic surgery position. Must be experienced in adult cardiac, general thoracic, and peripheral vascular surgery. Excellent fringe benefits. Malpractice paid.

Please direct curriculum vitae to Hawaii Permanente Medical Group, Inc, 3288 Moanalua Rd, Honolulu, HI 96819. An equal opportunity employer. 204J/L

Cardiovascular and thoracic surgeon wanted to join busy practice in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308. (Telephone: (305) 561-9781 after 9:00 PM.) 208J/C

A busy cardiothoracic group in the NYC area is looking primarily for a thoracic surgeon with potential for cardiac surgery in the future. Excellent salary and benefits. Respond with curriculum vitae.

Please respond to Box 211. 211J/L

Cardiothoracic surgeon needed to join rapidly expanding group in California's central valley. Prefer experienced individual with demonstrable skills in adult cardiac, thoracic, and peripheral vascular surgery. Generous salary with early partnership to right individual. Send curriculum vitae and references. Replies confidential.

Please respond to 216, The Annals of Thoracic Surgery, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010. 216JK

Cardiothoracic surgeon, board certified with 2 to 4 years experience in cardiac surgery, wanted to join a growing group of cardiac surgeons, cardiologists, and pulmonologists in the Pacific Northwest. General thoracic and vascular experience necessary. Research opportunities available, academic links possible. Salary leading to full partnership in 2 or 3 years; potential exists for leadership in major cardiac program.

Please send curriculum vitae to John R. Bauman, Administrator at The Thoracic Clinic, PC, 507 NE 47th Ave, Portland OR 97213-2282. 124C/L

Cardiothoracic surgeon, BE/BC, to join expanding practice of cardiac/thoracic surgery at 719-bed hospital (Rhode Island Hospital) and major affiliate of medical school. Excellent clinical and investigational facilities available with ample opportunities for clinical or basic science research. Private practice with academic affiliation, solid general surgical residency with active medical student participation. Salaried position leading to eventual partnership for right individual.

Send curriculum vitae to Arun K. Singh, MD, 110 Lockwood St, Providence, RI 02903; (401) 274-7546. 217K

Cardiothoracic/vascular surgeon, BE/BC, with primary interest in cardiac surgery, wanted immediately to replace member of established 6-physician surgical group practicing in Allentown, PA. Competitive salary/fringe benefits. Position leads to partnership for successful candidate. Please respond with CV, history of clinical experience, references, and salary requirements.

Please respond to Box 218. 218K/D

Cardiothoracic surgeon: Exceptional opportunity for an additional cardiothoracic surgeon to join an established, multispecialty group practice in the Tampa Bay area. Recent graduate preferred. Must be experienced in all areas of adult cardiac, thoracic, and peripheral vascular surgery. Training in valve repair, electrophysiologic surgery, and left ventricular assist devices desirable. Guaranteed first year salary leading to potential directorship.

For immediate consideration, rush CV in confidence c/o Recruitment Committee: Diagnostic Clinic, PO Box 2901, Largo, FL 34649. 219K/A

Cardiovascular and thoracic surgeon—Board certified with 5–10 years experience in cardiac surgery wanted to start new adult program in regional medical center in northwestern lower Michigan. This modern practice situation is located in renowned midwestern vacation area featuring sailing, skiing, hunting, fishing, and other outdoor recreational opportunities as well as excellent schools. Please send CV and references.

Please respond to Box 220. 220K/L

Cardiovascular and thoracic surgeon wanted for busy, established private practice. No cardiac surgery. Located in Great Lakes area. BC/BE. Please respond with CV and references.

Please respond to Box 221. 221K

Cardiovascular surgeon wanted to join busy private practice in excellent Florida location. Practice includes all aspects of adult cardiovascular and thoracic surgery. Would prefer a recent graduate. Send curriculum vitae.

Please respond to Box 223. 223K/A

Cardiothoracic surgeon wanted—to join established cardiac and thoracic surgeon in the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Please send curriculum vitae.

Please respond to Box 225. 225K/A

Cardiac-thoracic surgeon, needed for expanding program on west coast of Florida. Excellent opportunity, private practice, primarily adult cardiac including cardiac transplantation. BC/BE required. Please send CV.

Please respond to Box 226. 226K/D

Thoracic surgery residency: Newly approved residency in thoracic surgery to begin July 1990 at the University of Massachusetts Medical School/Hospital. Three-year program includes training in adult and pediatric cardiac and general thoracic surgery.

Interested candidates may contact Thomas J. Vander Salm, MD, Department of Surgery, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655. 228K/A

Physician's assistant with cardiovascular experience: Busy 3-man cardiovascular surgical practice needs a PA with CV experience. Computer experience and an interest in clinical research also required. Salary negotiable. Excellent benefits. Great location for person who enjoys outdoor recreation.

Contact Cardiothoracic Associates, PC, 2005 Franklin St, Midtown II, Suite 410, Denver, CO 80205; telephone (303) 832-6165. 229K

Texas Gulf Coast institution is planning a new state-of-the-art cardiovascular program. Seeking an aggressive, young, and experienced cardiovascular surgeon skilled in myocardial revascularization, valve repair and replacement, cardiac emergencies, and vascular surgery. Unique opportunity in a contemporary environment enjoying the full support of administration and established staff. Initial contact must include curriculum vitae and personal references.

Please respond to Box 230. 230KL

Cardiovascular surgeon, board certified or eligible, wanted to join surgeon to develop new program in the Midwest, outside Chicago. Experience in adult cardiovascular and thoracic surgery required. Strong interest in private practice and program development essential. Salary leading to partnership. Starting date negotiable. Personal interview a must. Please send curriculum vitae and record of personal operative experience.

Please respond to Box 231. 231K

Surgeon, cardiovascular/thoracic: The Santa Clara Valley Medical Center, San Jose, California, public hospital and a major teaching affiliate of Stanford University is looking for a full-time BC/BE cardiovascular and thoracic surgeon to perform open and closed cardiac and noncardiac thoracic operations, and to supervise and teach surgical house staff and medical students.

Please send CV to Maria Allo, MD, Chief of Surgery, Santa Clara Valley Medical Center, 751 South Bascom Ave, San Jose, CA 95128. Santa Clara Valley Medical Center provides competitive compensation and benefits and is an equal opportunity employer. 232K

Position available for associate cardiovascular and thoracic surgeon to first assist at open heart, pulmonary, and vascular operations, and care for patients in intensive care unit and perioperatively. Position requires an MD degree with a current California state license and a minimum of three years postgraduate training in surgery. Send curriculum vitae.

Please respond to Box 233. 233K

Pediatric cardiovascular surgeon wanted to join full-time academic staff. Applicants should be board eligible or certified in thoracic surgery with a special interest in pediatric cardiovascular surgery and research. Additional transplant experience would be favorable.

Applicants should respond with curriculum vitae to Frank M. Midgley, MD, Chairman, Cardiovascular Surgery, Children's Hospital National Medical Center, 111 Michigan Avenue, NW, Washington, DC 20010; telephone: (202) 745-2137. 234KL

Faculty position at Associate Professor level in congenital heart surgery for a major university children's hospital. Experience should include cardiac transplantation and complex neonatal reparative surgery. Position also requires substantial research and teaching activity. Send curriculum vitae, bibliography, and two letters of recommendation. The University of Pennsylvania is an equal opportunity/affirmative action employer.

Please respond to Box 235. 235K

Faculty position in cardiothoracic surgery for a major university medical center. Candidate must be interested in cardiac transplantation and immunobiologic research and satisfy UNOS criteria as transplant surgeon. Position requires a substantial research, clinical, and teaching commitment. Faculty rank commensurate with credentials. Send curriculum vitae, bibliography, and two letters of recommendation. The University of Pennsylvania is an equal opportunity/affirmative action employer.

Please respond to Box 236. 236K

Cardiovascular and thoracic surgeon: Position available for a board certified or board eligible to join practice in Northeast involved in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 227. 227K

SITUATIONS WANTED

Cardiovascular and thoracic surgeon, 42, American, ABS, ABTS, ABVS. Eight years private practice experience. Completed overseas fellowship in heart and heart/lung transplantation. Seeks opportunity with individual or group practice of cardiovascular and thoracic surgery. Curriculum vitae and references upon request.

Please respond to Box 199. 199G/K

Cardiothoracic surgeon, 36, wishes to relocate, preferably in Southeast or Midwest. ABS/ABTS. University and private group practice with extensive experience in complex adult thoracic and cardiovascular surgical disease. Interested in starting new program or associating with established practice.

Please respond to Box 177. 177JK

FELLOWSHIPS

Fellowship in thoracic and cardiovascular surgery at Baylor University Medical Center.

Contact Dr Harold C. Urschel, 3600 Gaston Avenue, Suite 1201, Dallas, TX 75246; telephone (214) 824-2503. 129G/L

Surgical fellow—Clinical fellowship position available in adult and pediatric cardiothoracic surgery. Large volume of cases. NJ license required. Excellent salary and benefits.

Please contact Lynn B. McGrath, MD, Department of Surgery, Deborah Heart & Lung Center, Browns Mills, NJ 08015. 167G/L

Thoracic-cardiovascular fellowship. Fellowship in Thoracic-Cardiovascular Surgery available immediately at Winthrop-University Hospital, Mineola, New York. This 533-bed hospital has ultramodern cardiac surgical facilities. Training includes a large variety of cardiac, thoracic and peripheral vascular cases.

Apply to M. Mohtashemi, MD, Chairman, Thoracic-Cardiovascular Surgery, Winthrop-University Hospital, Mineola, NY 11501. 206J/L

Fellowship in cardiovascular surgery for 1 year available January 1 or July 1 with group doing approximately 1,000 cardiac procedures per year and large volume of vascular procedures. ABS eligible or certified, California medical license required or readily obtainable. Ideal for a person between general surgery residency and thoracic surgery residency.

Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 222K/D

Cardiovascular and thoracic surgery fellow: Arizona license required.

Contact Cecil C. Vaughn, MD, 6036 N 19th Ave, Suite 405, Phoenix, AZ 85015; telephone (602) 242-1200. 224K

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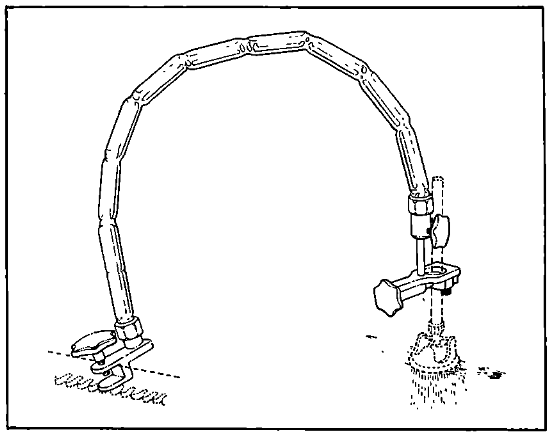

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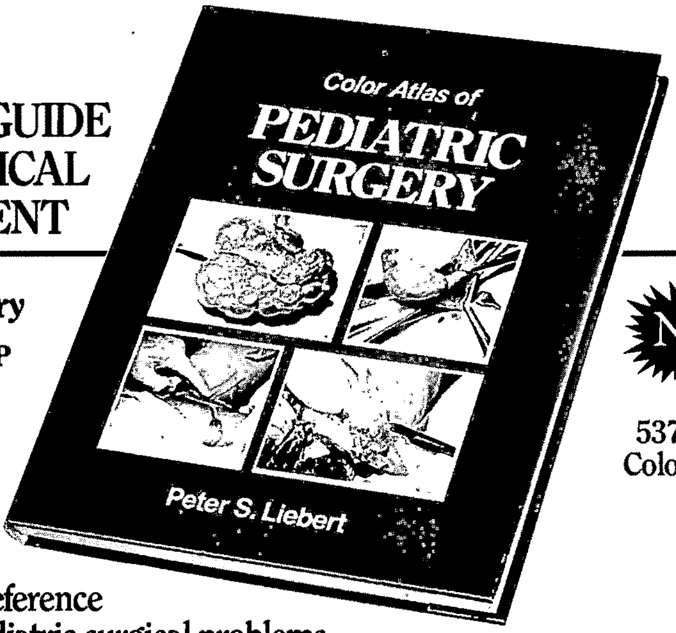

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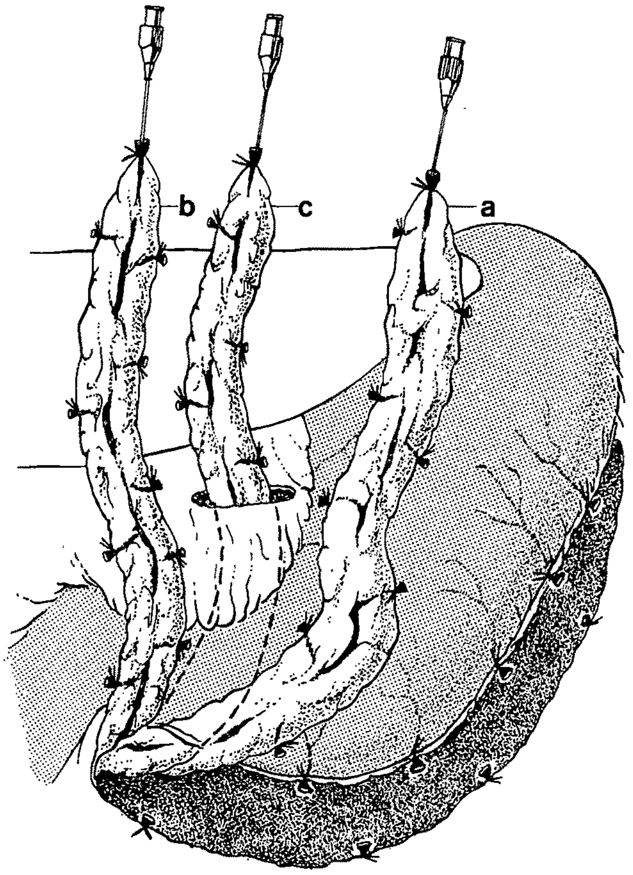

Figure 4. *Routes of the right gastroepiploic graft to the diaphragm are:* **(A)** *anterior to the stomach (rare),* **(B)** *anterior to the duodenum, and* **(C)** *posterior to the stomach through the avascular space in the lesser sac (most common).*

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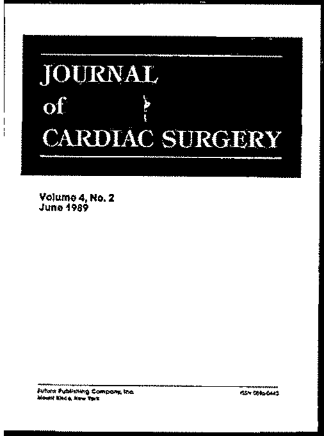

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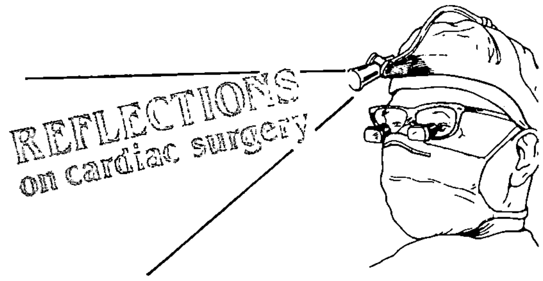

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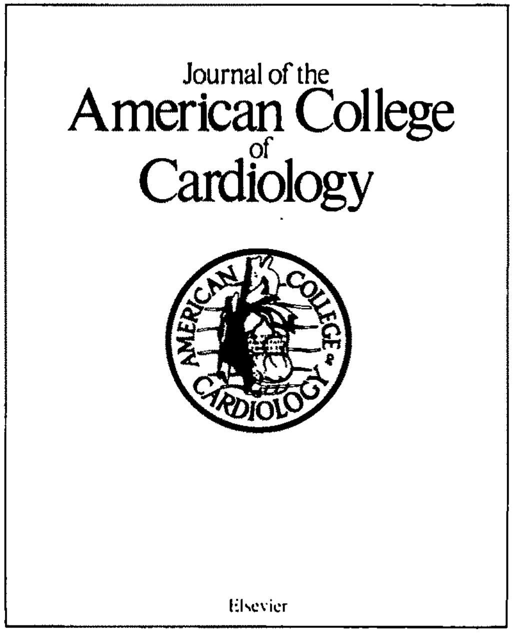

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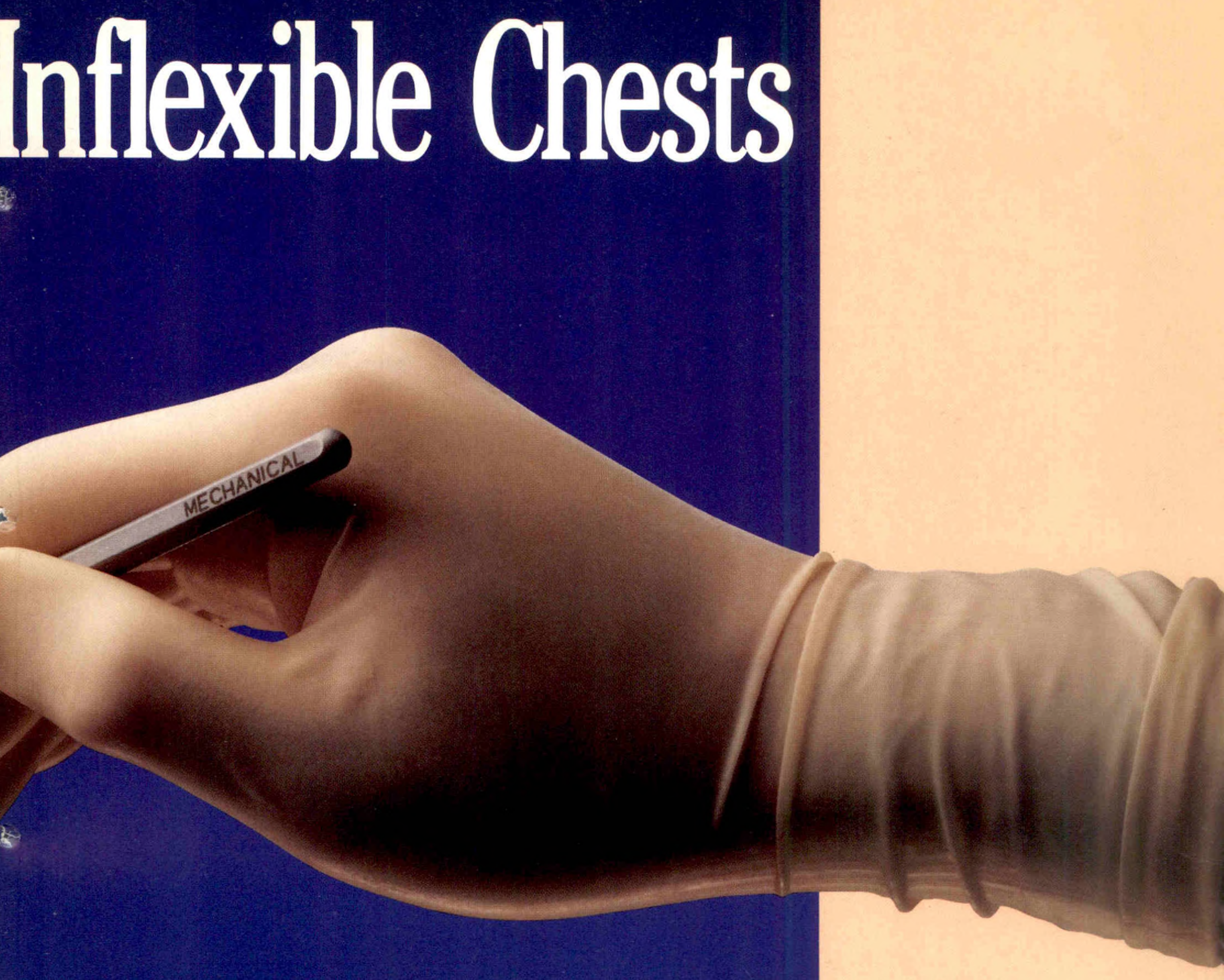

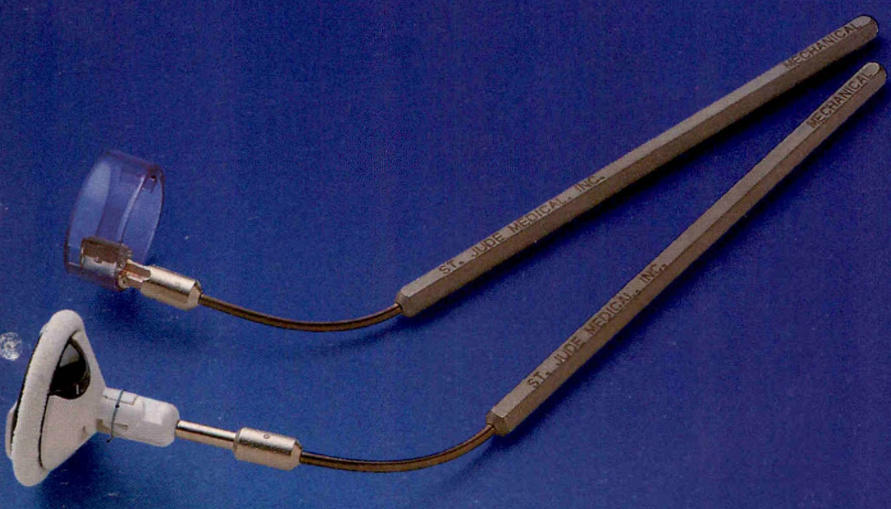

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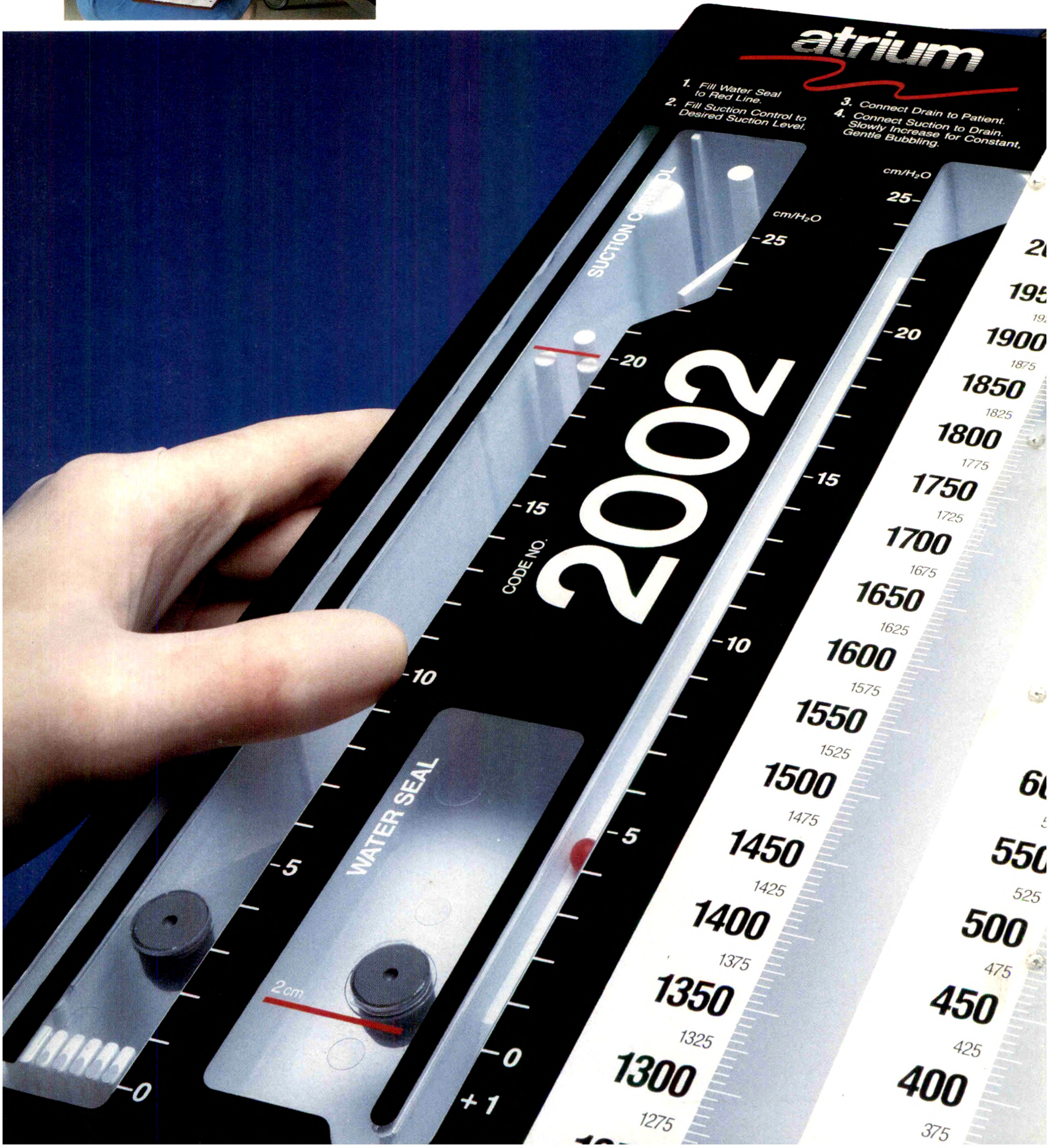
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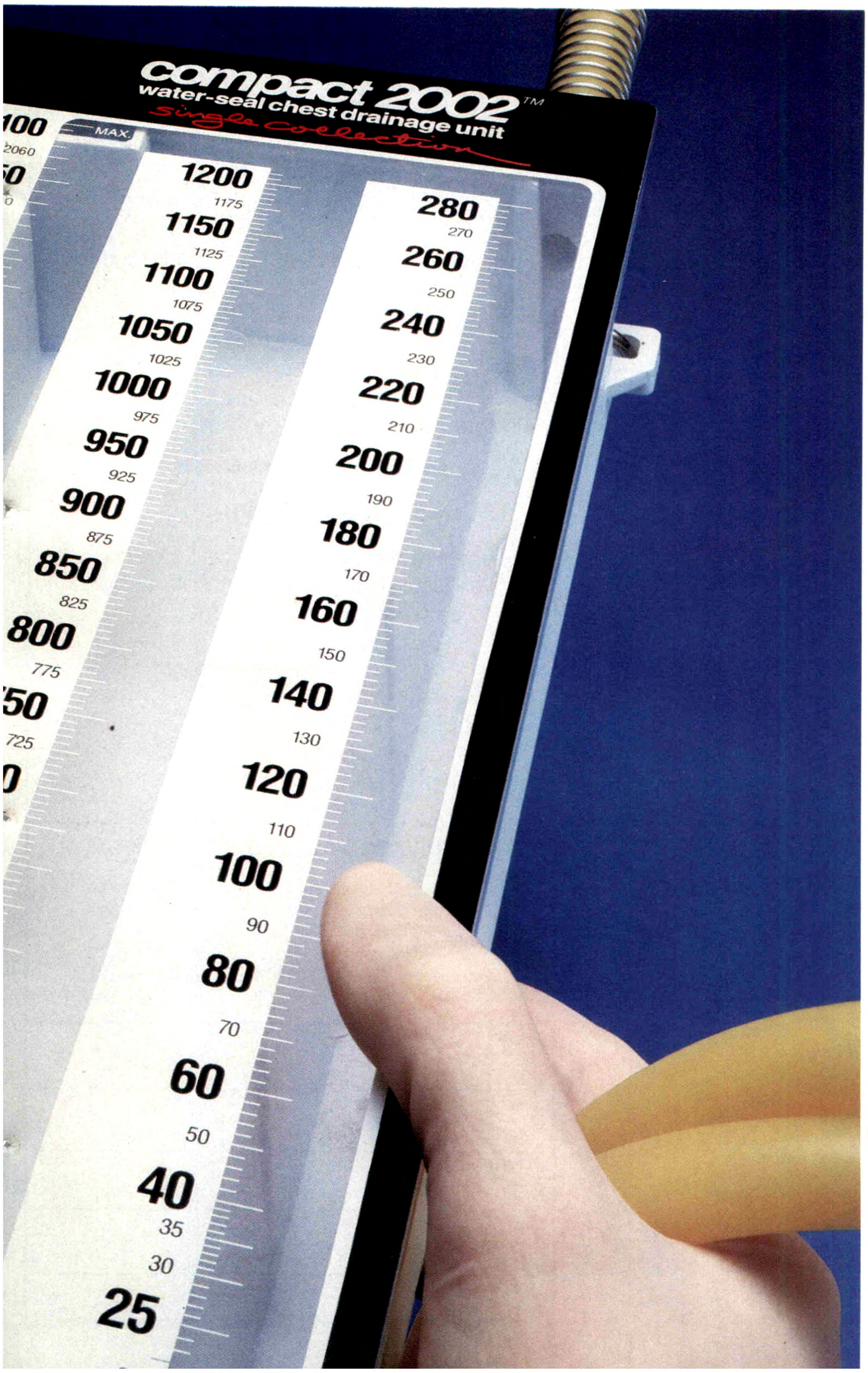

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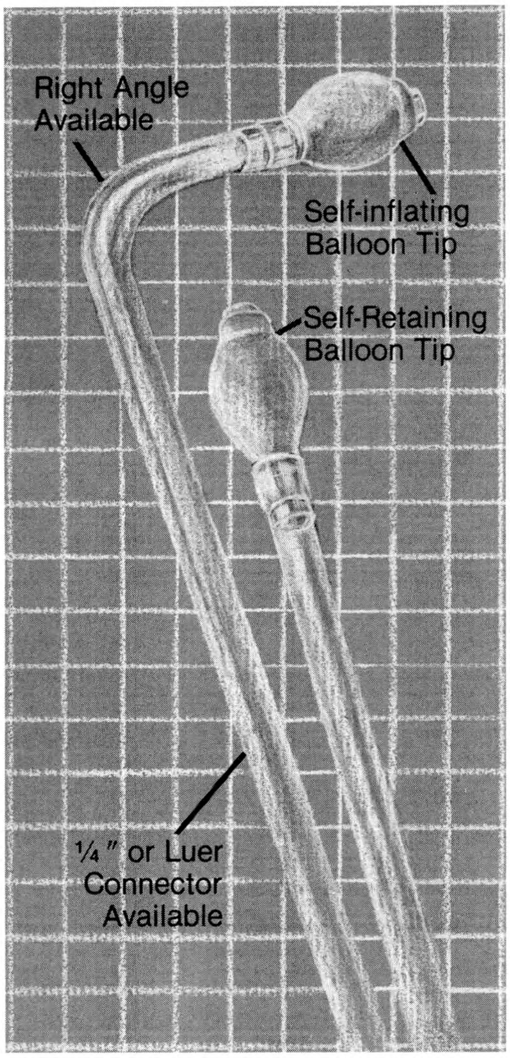
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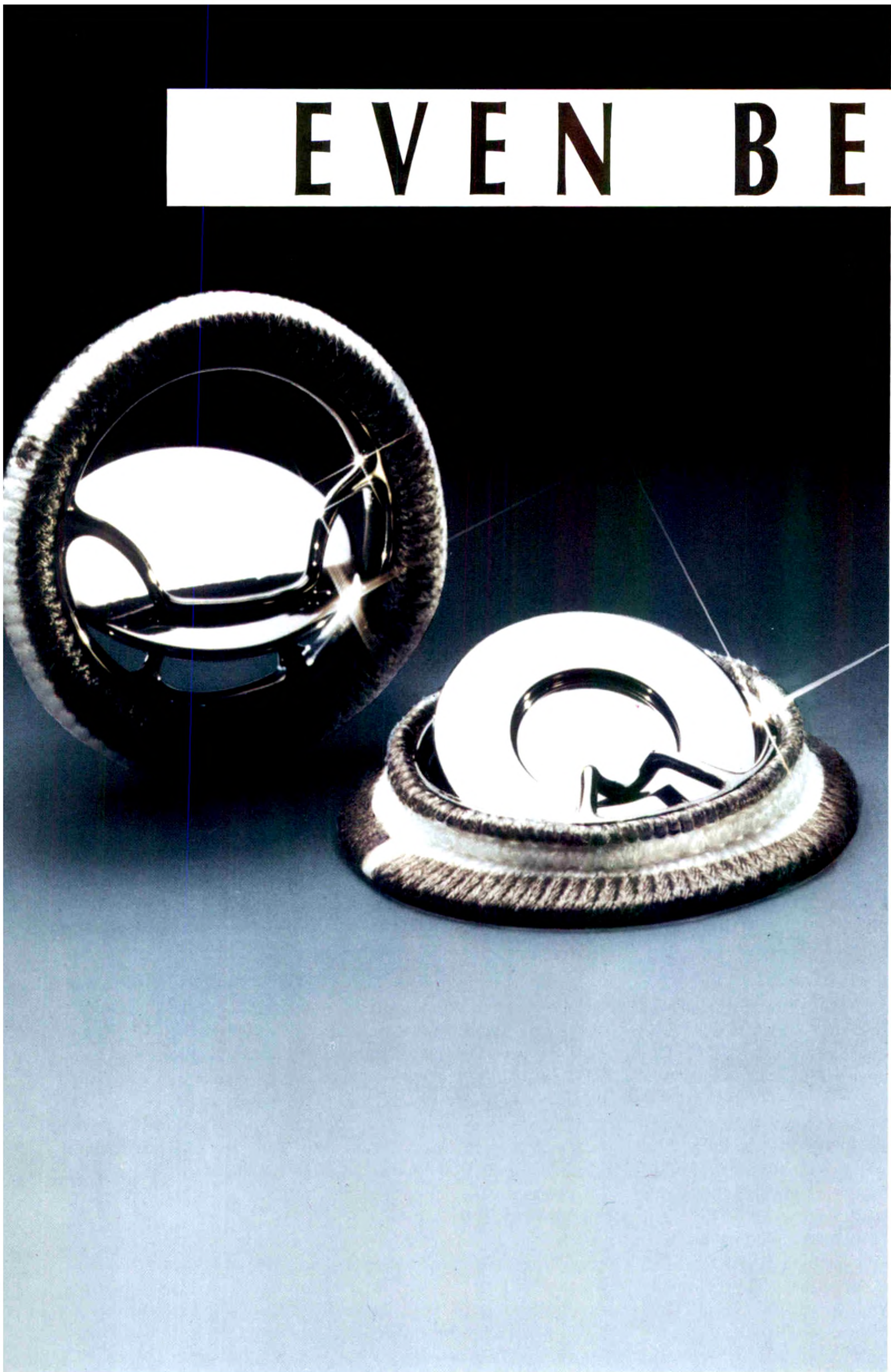

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Chapter in Book

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Original Articles

Cerebrospinal Fluid Drainage and Steroids Provide Better Spinal Cord Protection During Aortic Cross-Clamping Than Does Either Treatment Alone
Thomas T. Woloszyn, MD, Corrado P. Marini, MD, Matthew S. Coons, MD, Ira M. Nathan, PhD, Samir Basu, MD, Anthony J. Acinapura, MD, and Joseph N. Cunningham, MD

Direct Revascularization of Bronchial Arteries for Lung Transplantation: An Anatomical Study
Hans H. J. Schreinemakers, MD, Walter Weder, MD, Shinichiro Miyoshi, MD, Baron D. Harper, MD, Shinji Shimokawa, MD, Thomas M. Egan, MD, Robert McKnight, MD, and Joel D. Cooper, MD

Disruption of the Aortic Anastomosis After Heart-Lung Transplantation
Robert D. Dowling, MD, Naoum Baladi, MD, Marco Zenati, MD, J. Stephen Dummer, MD, Robert L. Kormos, MD, John M. Armitage, MD, Samuel A. Yousem, MD, Robert L. Hardesty, and Bartley P. Griffith, MD

Esophagectomy for Esophageal Disruption
Mark B. Orringer, MD, and Mack C. Stirling, MD

Evolving Patterns in the Surgical Treatment of Malignant Ventricular Tachyarrhythmias
John A. Elefteriades, MD, Lee A. Biblo, William P. Batsford, MD, Lynda E. Rosenfeld, MD, Richard W. Henthorn, MD, Mark D. Carlson, MD, Albert L. Waldo, MD, Jack Hsu, MD, and Alexander S. Geha, MD

Infusion of Nifedipine After Coronary Artery Bypass Grafting Decreases the Incidence of Early Postoperative Myocardial Ischemia
Rainald Seitelberger, MD, Werner Zwölfer, MD, Thomas M. Binder, MD, Sebastian Huber, MD, Friedrich Peschl, MD, Josef Spatt, MD, Severin Schwarzacher, MD, Christoph Holzinger, MD, Farid Coraim, MD, Heinz Weber, MD, and Ernst Wolner, MD

Primary Tracheal Tumors: Treatment and Results
Hermes C. Grillo, MD, and Douglas J. Mathisen, MD

Ten-Year Experience With Aortic Valve Replacement in 482 Patients 70 Years of Age or Older: Operative Risk and Long-Term Results
A. C. Galloway, MD, S. B. Colvin, MD, E. A. Grossi, MD, F. G. Baumann, PhD, R. Esposito, MD, G. H. Ribakove, MD, A. T. Culliford, MD, J. N. Slater, MD, E. Glassman, MD, S. Harty, RN, and F. C. Spencer, MD

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Arlen G. Fleisher, MD, G. Frank O. Tyers, MD, David Hu, MD, Eric M. Webber, MD, and Cameron Essery, MD

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A. J. B. Kirk, FRCS, I. D. Conacher, FFARCS, P. A. Corris, MRCP, T. Ashcroft, FRCPath, and J. H. Dark, FRCS

How to Do It

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J. A. R. Gaer, FRCS(Ed), C. Blauth, FRCS, E. R. Townsend, FRCS, and S. W. Fountain, FRCS

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Steven F. Bolling, MD

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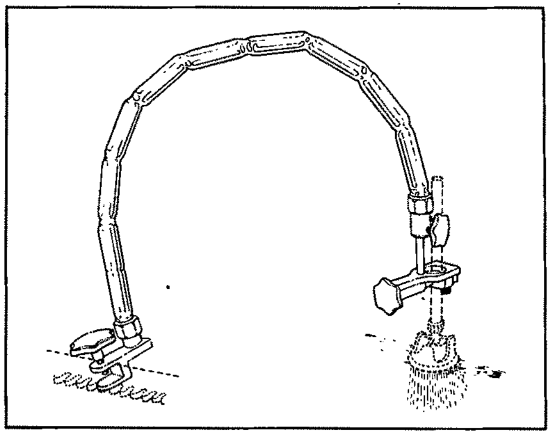
***Candida* Mediastinitis After a Cardiac Operation**
Donald D. Glower, MD, James M. Douglas, Jr, MD, James W. Gaynor, MD, Robert N. Jones, MD, and H. Newland Oldham, Jr, MD



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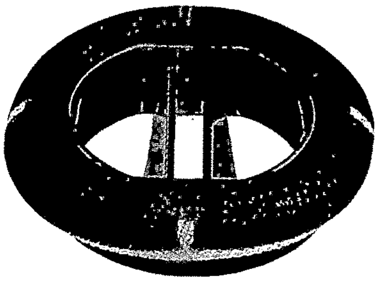

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STS Presidential Message to Membership

Activities in all areas of our Society's interests seem to have escalated so enormously during the past several months that increased communication to the membership is imperative. During my term in office, I will attempt to periodically transmit updates on Society activities to the membership and hope, in turn, to encourage input and comments from our members to the leadership.

One area requiring an enormous expenditure of effort and time addresses governmental affairs and, especially, physician reimbursement. Subcategories of this activity include:

1. Addressing proposed regulations;
2. Addressing decreased Medicare reimbursement of some so-called overpriced procedures (including coronary artery bypass);
3. Addressing the resource-based relative value scale (RBRVS; the Hsiao study);
4. Appraising "expenditure target" regulations; and
5. Assessing proposed activities concerning practice guidelines and results research.

We (joint effort between The Society of Thoracic Surgeons, The American Association for Thoracic Surgery, The Western Thoracic Surgical Association, and the Southern Thoracic Surgical Association) have been able to effect modest relief from excessive reduction in Medicare reimbursement for coronary artery bypass, valve replacement, and some pulmonary resective procedures. We (again, the four major thoracic surgery societies) have received the endorsement of key governmental agencies to be restudied for our portion of the RBRVS by a respected group other than Hsiao and associates because of acknowledged flaws in the first RBRVS survey of thoracic surgery. George Miller and the Ad Hoc Committee on Physician Reimbursement have contributed an inestimable number of man-hours in efforts to favorably influence legislation coming before Congress in the near future and over the next several years. Our Chicago Headquarters Staff and our legislative consulting group in Washington (Health Policy Alternatives) have provided invaluable support in these and other efforts.

Scientific programming will always have the highest priority in Society activities; this includes the Annual Scientific Program, the Postgraduate Course, publication of *The Annals*, continuing education, educational resources, and other programs such as symposia that may be held from time to time.

Because of the increasing demands that accompany such activities, The Society must address the issue of membership. Our membership numbers have reached a plateau and this year, for the first time, the number of active members advancing to Senior status is greater than the number of new members. I solicit help from all of the membership to recruit eligible candidates who are known to you in your communities, in your teaching programs, and otherwise. Membership applications and staff assistance in processing are readily available from our Chicago office. Increased membership means more financial support needed for our broad range of interest, more involvement to help in committee deliberations, and increased ease of communications at the local and regional levels.

Examples of ways that individual members can be highly useful and effective include contacts with key political figures (sometimes far more effective at the local one-on-one level) and making key hospital administrative and medical staff personnel aware of the thoracic and cardiovascular viewpoint in such issues as extracorporeal supported angioplasty (Should cardiopulmonary support systems be initiated by cardiologists without surgical presence? Should perfusionists work with cardiologists without surgical presence? Should pump manufacturers promote use of cardiopulmonary support systems by cardiologists and market sales techniques directly to hospital administrators without cardiac surgical input? Should nurses or technicians not trained in perfusion techniques replace perfusionists in operating cardiopulmonary support systems or extracorporeal membrane oxygenators?). The Society cannot mandate action on issues such as are listed above; however, Society members, either as individuals or in groups, may be extremely effective at the local hospital level.

Because of the great scope of activities of The Society and the rapidity with which changes occur, it might help to speed communication if I were to visit as many local thoracic surgical society groups as possible over the next 12 to 14 months. I welcome your thoughts in this particular area and would be happy to visit any group directly if this would help in our deliberations. Sometimes communications can be far more effectively transmitted in groups such as this than by the written word, which is sometimes delayed in its distribution.

Many other topics will arise from time to time that require communication to the membership. I will try to forward this information to you on a periodic basis.

W. Gerald Rainer

W. Gerald Rainer, MD
President, The Society of Thoracic Surgeons

Early Bypass Operation After Acute Myocardial Infarction

John L. Ochsner, MD

Department of Surgery, Ochsner Clinic and Alton Ochsner Medical Foundation, New Orleans, Louisiana

In the past, multiple reports have explicitly demonstrated the dangers of general anesthesia and any major operation within 6 months after an acute myocardial infarction [1–5]. The shorter the interval between infarction and operation, the greater the hazards of reinfarction. Patients operated on within 6 months after infarction had a 30% to 45% reinfarction rate. This rate remained high but decreased to 15% to 20% in the 3- to 6-month interval and stabilized at 5% after 6 months. In addition, the mortality from reinfarction in these patients increased to 50%. Not only the infarcted myocardium but also the remaining viable muscle functions suboptimally in the early postinfarction period. Thus, elective surgical procedures were deferred for 6 months after an acute myocardial infarction and only life-threatening emergencies were considered for operation. This philosophy became a well-entrenched surgical axiom. Hence, in the early days of myocardial revascularization, cardiac surgeons naturally adhered to those principles learned in general surgery. However, most of us encountered the frustrating experience of watching patients' infarctions extend or recurrent infarctions develop during this postinfarction observational period. Postinfarction extension also has a mortality two to five times greater than the mortality after a primary infarction [6, 7].

Because overall mortality in the first year after myocardial infarction ranges between 10% and 15%, with more than one third of the deaths occurring in the first 6 weeks and one half of the deaths occurring in the first 3 months, it appears rational to perform an early operation to correct any underlying lesions causing ischemia or those with the potential of producing a catastrophe. In addition, assuring the best preoperative condition of the patients with an acute myocardial infarction by progressive invasive monitoring of hemodynamic status and prompt treatment of any hemodynamic aberrations can decrease perioperative mortality and morbidity [8].

The initial experience with early postinfarction surgical revascularization was generally disappointing; however, in recent years there has been a renewed interest in preventing extension of the infarction or new infarctions from a remote area, thus preserving myocardial mass and left ventricular function. In 1983, Roberts and co-workers [9] reported a series of 20 patients with postinfarction angina who were managed medically for a mean interval of ten days before surgical revascularization; there were no operative deaths in the series. Based on this experience, Roberts and co-workers [9] suggested that aggressive medical stabilization should be undertaken before coronary artery bypass grafting is performed. Since then, many studies have shown that the timing of postinfarction revascularization was unimportant and that the interval between infarction and revascularization was not a predictor of postoperative cardiac failure [10–15]. These studies show that the risk of operation is no greater than that in elective cases in which the patient has stable angina or no angina, that the risk is slightly increased in patients who have angina at rest, the risk is substantial in patients with angina requiring intraaortic balloon counterpulsation for control of pain, and it is catastrophic in patients with severe postinfarction ischemia or extension complicated by cardiogenic shock (50%). Hence, one must question the indication for stabilization attempts that delay operation until a safe interval has elapsed; here lies the controversy.

As elsewhere in medicine, the decision to perform an operation is based on factors relating to the individual patient. Today, almost all patients who have a myocardial infarction undergo coronary angiography to visualize the coronary anatomy to determine if other myocardium is in jeopardy and ventriculography to determine the function of the myocardium. The American College of Cardiology recognized that the indications for coronary angiography and its timing after acute myocardial infarction change as experience evolves and have set different criteria for three different phases: (1) evolving myocardial infarction, (2) completed myocardial infarction, and (3) convalescent myocardial infarction [16]. Most cardiologists believe that all patients with non–Q-wave infarction should undergo early coronary angiography because they have an increased risk of myocardial infarction at a later date and a high late mortality [17]. Also included in early coronary angiography are patients with Q-wave infarction who (1) are symptomatic, (2) are young (ie, aged less than 50 years), and (3) have a positive low-level treadmill test before discharge from the hospital. If the low-level treadmill test is negative, the patient should return in 4 to 6 weeks for a maximum stress test. If the maximum stress test is positive, the patient should undergo coronary angiography. Thus, a deliberation based on the patient's age, physiological state, coronary anatomy, and myocardial function allows proper decision as to early revascularization after an acute myocardial infarction.

In this issue, Floten and associates [18] have demonstrated that the long-term results after early surgical revascularization of patients with acute myocardial infarction are not different from results of patients who undergo operation and who have had a remote infarction. Accordingly, Floten and associates accurately state that there

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appears to be little to gain by postponing coronary artery bypass grafting. This study was performed between 1974 and 1987, and new therapeutic directions have evolved during and since that period. Today, the preferred treatment for postinfarction patients who are candidates for revascularization is reperfusion by percutaneous transluminal angioplasty (PTCA). Occasionally, bypass grafting is appropriate when the site, extent, and characteristics of the offending coronary lesions are undesirable or hazardous for PTCA. In the study by Floten and associates, 28% of the patients were operated on for only one or two diseased vessels. At present in most centers, such individuals would be treated with PTCA. Many patients with three-vessel disease probably also would be treated initially with PTCA, particularly those who have a definable "culprit" symptomatic lesion. Thus, today a substantial number of patients in the study would undergo PTCA as their initial form of therapy. If one reviews the patients with information referable to the number of vessels involved (Table 2 in the study by Floten and associates) and excludes those with one- or two-vessel disease (who surely today would be treated with PTCA), the operative mortality increases from 4.1% to 5.5%. Even though assignment of the low-risk patients to a different treatment would theoretically raise the operative mortality, Floten and associates report that the true operative mortality in this series also changed with time from 6.7% before 1981 to 4.4% after 1981.

When surgeons elect to postpone operation on postinfarction patients until a later date, they are selecting the more stable candidates for myocardial revascularization. However, to affect the hemodynamics of a patient with acute myocardial infarction and to prevent further loss of myocardium or death in the postinfarction period, surgeons must operate early. Unfortunately, patients who are not amenable to PTCA and patients who fail PTCA are usually the poorer candidates or unstable patients and consequently at greater risk.

In the study by Floten and associates, long-term survival was significantly affected by the patient's age and left ventricular end-diastolic pressure, which one would expect. Although postinfarction angina and extent of anatomical disease detrimentally influenced the operative mortality, these factors did not affect long-term survival.

Although this study excludes patients in cardiogenic shock, other studies have shown that early surgical revascularization has a great advantage over conventional medical therapy, thus giving further support to early surgical intervention [12, 14].

The goals in treating acute myocardial infarction are to prevent death and loss of myocardium, to maintain ventricular function, and to improve the patient's quality of life. Because surgical revascularization can be accomplished in the early postinfarction period without increased risk and because long-term results are equal to results of operations performed for patients with remote infarction, coronary artery bypass grafting should continue to play a definitive role in the treatment of select patients after myocardial infarction.

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Cerebral Blood Flow During Cardiopulmonary Bypass: Some New Answers to Old Questions

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The questions of adequate brain blood flow and preserved neurological function are as old as cardiopulmonary bypass (CPB) itself. John Gibbon [1] realized in the beginning "it is obvious that any operative procedure upon the heart could be performed better if that organ were temporarily relieved of its function of pumping blood," but the method had to prevent "irreparable neurological changes."

Although the development and success of the modern pump-oxygenator in clinical practice has been truly remarkable, advances in cerebral protection during cardiac operations have not come as quickly nor gone as far as those in myocardial protection. As perioperative death (usually cardiac in origin) has declined over time (even in high-risk patient populations) [2] postoperative neuropsychological dysfunction has emerged as an increasingly important aspect of cardiac surgical morbidity. Postoperative neuropsychological dysfunction is devastating to the patient, family, and physicians alike, especially after a perfect cardiac operation. In fact, it has recently been stated in the preface to a monograph on the subject [3] that "brain injury is one of the most unacceptable complications sustained during heart surgery."

It is generally considered, although it has not been proved, that neurological injury is caused by inadequate cerebral blood flow whether it be hypoperfusion or embolic in origin. Regardless of the cause, those episodes of neuropsychological dysfunction that result from irreversible ischemia have permanent sequelae, whereas those that are transient represent a stunned brain syndrome that improves over time. Understanding cerebral blood flow during CPB should provide answers to causes of inadequate tissue perfusion. It is only through investigations such as the one in this issue by Johnsson and co-workers [4] that we can obtain answers to the fundamental physiological questions concerning cerebral blood flow during CPB.

Johnsson and colleagues [4] report on cerebral blood flow measurements during cardiac operations using xenon 133 washout methodology. This type of measurement, determined from a relatively large region of the brain, reflects global cerebral blood flow (CBF) and can be regarded as hemispheric, as distinguished from regional cerebral blood flow (CBF_r) in a localized part of the brain that might be measured with radiolabeled microspheres or use of positron emission tomographic scan. Regional cerebral blood flow methods are not applicable to humans in the operating room. The ^{133}Xe CBF measurements are feasible in the operating room and are appropriate to answer the question posed by Johnsson and co-workers: Is the cerebral vasculature sensitive to arterial CO_2 tension ($PaCO_2$) during moderate hypothermia? Global cerebral blood flow is responsive to $PaCO_2$ before, during, and after cardiopulmonary bypass, with or without moderate hypothermia. As Johnsson and co-workers point out this report is confirmation of the recent work of others [5–8], and this phenomenon was first reported by Wollman and co-workers [9] in 1966 using a Fick arteriovenous O_2 method. The importance of the observation is that, despite the enormous physiological abnormalities wrought by cardiopulmonary bypass, the cerebral circulation acts "normally," ie, dilates in response to increased $PaCO_2$.

As many of the determinants of normal brain perfusion become externally controlled during CPB (eg, flow rate, perfusion pressure, temperature, $PaCO_2$), knowledge of the effect of these variables on CBF during CPB is essential. Recent ^{133}Xe CBF studies have enabled several investigative groups to provide new information on these questions regarding CBF and CPB (Table 1). We now know that during CPB temperature, anesthetic depth, cerebral oxygen consumption ($CMRO_2$), and $PaCO_2$ are the major factors that regulate CBF during CPB. Factors that do not influence CBF (within the usual bounds of common practice) are blood pressure, pump flow, and age ($\leq$65 years). The reason that blood pressure ($\geq$30 mm Hg) and pump flow ($\geq$1.6 L/min/m^2) do not affect CBF is that the brain is able to autoregulate; changes in cerebral vascular resistance increase or decrease to change flow in response to metabolic need during variations in perfusion pressure or pump flow rate. Decreased CBF occurs with anesthesia and lowered temperature because the $CMRO_2$ is reduced and the brain requires less oxygen delivery (CBF). Thus, the larger question than is the brain vasculature reactive to CO_2 is whether or not cerebral blood flow autoregulation remains intact during CPB. This question has been answered in the affirmative. The fact that cerebral autoregulation is preserved under the usual conditions of CPB explains why the brain receives adequate perfusion in most instances despite the very low perfusion pressures often encountered.

Although autoregulation is preserved under most circumstances of CPB, there are conditions under which this is not true and patients in whom this is not so. Autoregulation during CPB is lost with profound hypothermia ($\leq$22°C) [6] and when pH-stat blood gas management is used [8], meaning that CBF is dependent on pump flow and perfusion pressure. It is possible that autoregulation

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Table 1. Recent Knowledge Concerning Factors Affecting Global Cerebral Blood Flow During Cardiopulmonary Bypass[a]

Variable	CBF Relationship
$CMRO_2$	Direct
Anesthetic depth	Direct
Temperature	Direct
$PaCO_2$	Direct
PaO_2	Inverse
Pump flow ($\geq$1.6 L/min/m^2)	None
Mean blood pressure ($\geq$30 mm Hg)	None

[a] During nonpulsatile flow in patients managed with alpha-stat blood gas strategy (see text) and without neurological problems or hypertension. These relationships suggest that cerebral blood flow autoregulation is preserved during cardiopulmonary bypass with alpha-stat blood gas management.

CBF = global cerebral blood flow; $CMRO_2$ = global cerebral oxygen consumption; $PaCO_2$ = arterial carbon dioxide tension; PaO_2 = arterial oxygen tension.

Sources: references 5–11.

is impaired or lost in patients with diabetes, untreated hypertension, and certain neurological disorders, and with some anesthetics. Loss of autoregulation will require changes in customary practice; for example, perfusion should be at higher flows, perfusion pressure should be maintained at higher levels, and possibly pH-stat blood gas management should be used.

The fact that cerebral vasculature is responsive to $PaCO_2$ in most patients during customary CPB inevitably has sparked a discussion, reminiscent of the carotid endarterectomy/ventilation debate, whether to hyperventilate, hypoventilate, or normoventilate patients to change the $PaCO_2$ in an effort to manipulate the CBF. The general conclusion of that debate has been to keep patients eucapneic to avoid cerebral steal physiology and to let the brain autoregulate. Johnsson and co-workers use this same intuitively pleasing argument (preservation of autoregulation) to recommend alpha-stat* blood gas management during CPB rather than pH-stat. pH-stat management increases CBF because $PaCO_2$ is higher with pH-stat than with alpha-stat management. Johnsson and associates state in their paper that using alpha-stat blood gas management prevents "cerebral hyperemia," and Murkin and colleagues [8] have shown that use of alpha-stat maintains a physiological coupling of CBF and $CMRO_2$; ie, CBF matches the $CMRO_2$. One is tempted to conclude as do Johnsson and associates that "alpha-stat management is the method of (blood gas management) choice" for hypothermic CPB. The presumption is that because CO_2 reactivity and CBF–$CMRO_2$ coupling are preserved during alpha-stat management and these are lost when pH-stat management is performed (because the cerebral vasculature dilates maximally or near maximally due to the CNS acidosis), this blood gas strategy is best. Relatively rapidly over the years of CPB use, alpha-stat management has been almost universally adopted probably because of its simplicity (because CO_2 is rarely added to the fresh gas supply to the oxygenator). Now there is a physiological basis for this practice, but unfortunately there are no neurological outcome data supporting the superiority of either blood gas management approach during CPB.

The work of Johnsson and co-workers, as well as that of others, does raise new questions regarding CBF and cardiac operations. The first question relates to the limitation of the ^{133}Xe methodology, which is appropriate for measure of CBF but not appropriate for CBF$_r$, where localized flow can be reduced by embolism or atheroscleroses with or without spasm. It is unlikely that embolic causes of neuropsychological dysfunction will be detected even with use of multiple detectors over various regions of the brain. Another limitation of this and other studies has been the exclusion of patient populations of great interest, for example, those with previous neurological symptoms (eg, transient ischemic attack or stroke): these excluded patients are more vulnerable to postoperative neurological injury. Also, most studies have excluded the very elderly and untreated hypertensive patients who, together with diabetics, are likely to have impaired cerebral autoregulation despite alpha-stat blood gas management. Finally, most of the scientific emphasis has been on measurement of CBF and answering the questions of whether cerebral vascular reactivity and cerebral autoregulation are preserved. These were important logical first questions to answer, but now more complex and important problems need to be addressed. It is, after all, not just CBF but the appropriateness of oxygen supply to demand that is crucial. Johnsson and co-workers have begun to look at this with the calculation of oxygen delivery, but it is really the delivery to utilization ratio (CBF/$CMRO_2$) that is of greatest concern even during hypothermia. This question can be answered and must be coupled with sensitive measures of neuropsychological outcome before we have the necessary information regarding the many questions concerning CBF and CPB. Then we can answer a vital question: To what degree does impairment of autoregulation (regardless of its cause) contribute to postoperative neuropsychological dysfunction? With greater understanding of the mechanisms of CBF and cerebral metabolism regulation, progress will surely come toward better cerebral protection during cardiac operations.

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*Blood gas management may be of two types: alpha-stat or pH-stat. Alpha-stat employs no temperature correction; ie, hypothermic specimens are interpreted as if they are normothermic. pH-stat employs temperature correction of the value to the hypothermia temperature of the patient. If $PaCO_2$ is 40 mm Hg with alpha-stat at 28°C, the $PaCO_2$ is 40 mm Hg at normothermia, whereas if $PaCO_2$ is maintained 40 mm Hg at hypothermic conditions using pH-stat, the $PaCO_2$ is approximately 60 mm Hg at normothermia (50% greater than alpha-stat or relatively hypercarbic, which causes cerebral dilation and impairment of "normal" autoregulation).

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It Is Time for a National Cardiothoracic Surgical Data Base

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The Society of Thoracic Surgeons will soon sponsor an opportunity for its members to participate in a national clinical data base system for cardiac and thoracic operations and outcome. This effort by the STS to establish a national data base is more than 5 years old. An ad hoc committee initiated a pilot program that, unfortunately, did not evolve to national scope. The development of small portable computers that were fast and interactive and had large storage capabilities has made possible the formation of many local data bases for cardiac surgery. The time is now ripe for those with existing data bases and those who have none at present to participate in this national effort.

The reasons for the initiation of this program by the STS are many. The formation of a group to undertake the effort was prompted by the release by HCFA (Health Care Financing Administration), a component of the Department of Health and Human Services, of raw mortality data for Medicare patients undergoing coronary artery bypass grafting procedures without respect to any of the then-known risk factors associated with patients during coronary artery disease.

The Standards and Ethics Committee of the STS released its "Statement of Concern" with approval of Council late in 1986, which was well received by practitioners and hospitals. The Standards and Ethics Committee at the Toronto meeting of the STS in September of 1987 developed a proposal for the Council for the development of a national data base system sponsored by the STS.

The pressing need was clearly evident from a number of facts. First, the experience of the Liability Committee of the STS had demonstrated that misrepresentation and misconception of operative risks were leading causes of malpractice actions. Second, the experience of the Standards and Ethics Committee of the STS had shown that neither the hospitals, the cardiologists, nor, in some cases, the surgeons had recognized the changing patterns of patient populations receiving coronary artery bypass grafting procedures, especially with the advent of catheter intervention, thrombolysis, and revascularization in the setting of acute myocardial ischemia. Hence, the prevalent professional and public perception that coronary artery operations carried little or no risk was perpetuated. A third driving force was the action of the JCAHCO (Joint Commission on Accreditation of Health Care Organizations). Quality assurance programs for every segment of the health care system were required. Recredentialing on a yearly basis was to occur based on the previous year's clinical performance by the practitioner. Clearly, results of the surgeons were going to be readily evident in contrast to those of psychiatrists. Finally, Congress required of HCFA and a suborganization, the PPRC (Physicians Payment Review Commission), a complete study and review of physicians' billings and payments under the Medicare system with the requirement of reporting the results in late 1989 and early 1990. The purpose was to decrease the rate of increase in health care costs, which have been substantially exceeding the inflationary rate for more than a decade. The initial report by the investigator (Hsiao) to the HCFA recommended a rearrangement of payment schemes with greater weight given to so-called cognitive skills than to technical ones. Cardiothoracic surgeons were to receive a reduction in payments of 25% to 30%. The need, therefore, to determine accurately the amount of services provided in toto by the cardiothoracic community had become acute.

Edmunds and Kaiser, in their preface to the report of the Committee for the state of the art symposium on coronary arterial surgery, made a pertinent strong plea.

> Proper solution to the risk-benefit equation requires knowledge of the natural history of the disease and of the incremental risk factors that affect operative mortality and long-term results. The surgeon must provide these data for the patient, the family and the cardiologist. Risk stratification is the essence of responsible cardiac surgery [1].

Ebert, in his keynote address to the Conference, stated:

> We, as a profession, should demand that data only be released based on patient classification. If the profession is not willing to develop a classification system that is simple enough to be understood by the public, then we cannot complain if data continue to be released in a somewhat questionable manner [2].

The STS thoracic and cardiac national data base will be initiated this fall with ten to 30 centers participating in the initial start-up. Approximately 50,000 cardiac cases will be available for comparison initially and an equal number will be added to the registry in the last quarter of 1989. Adult thoracic and cardiac surgical cases will be accepted initially. The campaign for all cardiothoracic surgeons to join as subscribers will begin in January 1990. The system will be highly secure so that the identity of each surgeon and patient is protected and available only to the subscrib-

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Dr Clark is the chairman of The Society of Thoracic Surgeons Ad Hoc Committee to Develop a National Database for Thoracic Surgery; the members are Anthony J. Acinapura, Richard P. Anderson, L. Penfield Faber, Frederick L. Grover, William R. E. Jamieson, Forrest L. Junod, Constantine Mavroudis, Richard B. McElvein, Richard M. Peters, and Quentin R. Stiles.

ing surgeon. National data will be lumped and not segregated by city or state. The local system will generate a quality assurance report for each surgeon ad lib. It is highly interactive and user friendly and has strong biostatistical and graphic capabilities. It has *no* business capabilities. Importantly, it carries an automated risk stratification system such that each surgeon can compare local patient subsets with the national experience. For the first time, we may be able to compare apples with apples.

The International Transplant Registry and the National Ventricular Assist Device Registry have kindly agreed to participate with inclusion of their respective data bases. The American Association for Thoracic Surgery, the Southern Thoracic Surgical Association, and The Western Thoracic Surgical Association have lent their organizational support to the effort.

We are on the brink, for the first time, of a voluntary national effort to gather, collate, and analyze the cardiac and thoracic surgical experience in the United States. Although the efforts have been substantial, the dividends to the individual cardiothoracic surgeon will quickly eclipse these and provide a new perspective and reality to the profession, the public, the media, and the government.

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Long-Term Survival After Postinfarction Bypass Operation: Early Versus Late Operation

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A study of 832 patients operated on within 30 days of infarction from 1974 to 1987 has resulted in 2,388 patient-years (maximum, 14 years) of prospectively acquired follow-up. This study excludes 74 patients in whom cardiogenic shock was the indication for operation. Five-year survival (± standard error) was 84% ± 2%, 85% ± 1%, and 90% ± 1%, and 10-year survival was 71% ± 4%, 68% ± 1%, and 78% ± 1% for patients with acute infarction, remote infarction, and no previous infarction, respectively. Age and left ventricular end-diastolic pressure significantly affected long-term survival for patients with acute infarction by both univariate and multivariate analysis. For patients aged less than 65 years, the 5-year and 10-year actuarial survival rates were 89% ± 2% and 80% ± 4%, compared with 75% ± 3% and 58% ± 9%, respectively, for patients aged more than 65 years. The survival percentages were 89% ± 2% and 75% ± 6% for patients with left ventricular end-diastolic pressure less than 15 mm Hg compared with 77% ± 5% and 67% ± 7% for patients with left ventricular end-diastolic pressure greater than 15 mm Hg. Operative mortality was 7.6% for patients operated on within 24 hours, compared with 4.1% for patients operated on between 2 and 30 days after infarction. Ten-year survival was similar (about 70%) for all timing groups. Based on these long-term results, there appears to be little to gain by delaying coronary artery bypass grafting, when indicated, after infarction occurs.

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Controversy persists regarding the relative risk and appropriate timing of surgical revascularization during the postinfarction period. Some investigators believe that it is appropriate to stabilize the patient medically and delay coronary artery bypass grafting for at least 1 month. Others believe that operation should be performed on an urgent basis to appropriately treat patients who are at high risk of infarct extension. In an attempt to address this controversy, we compared the early and long-term outcome after coronary bypass grafting in patients with acute myocardial infarction with the outcome of patients with a remote infarction and those who had suffered no previous infarction.

Material and Methods

From 1974 to 1987, 7,912 patients underwent coronary artery bypass grafting (CABG) at St. Vincent Hospital and Medical Center (Fig 1). Eleven percent of the patients were operated on within 30 days of acute myocardial infarction, 36% had a remote infarction (occurring more than 30 days before operation), and 53% had no history of infarction. This study excludes 74 patients in whom cardiogenic shock (systolic blood pressure less than 90 mm Hg and cardiac index less than 1.8 despite intraaortic balloon pump support) was the indication for operation.

The mean age for the patients with acute infarction was 61.4 years, ranging from 31 to 85 years (Table 1). Between 1974 and 1987, the mean age of the patients has increased from 52 to 63 years. The total follow-up was 2,388 patient-years (mean follow-up, 2.9 years), and follow-up was 93% complete during 1986 and 1987.

Since 1974, an increasingly greater percentage of patients has been operated on for acute infarction (Fig 2). Before 1981, 2% to 5% of CABG patients were operated on for acute infarction. This percentage has increased to 20% since 1985.

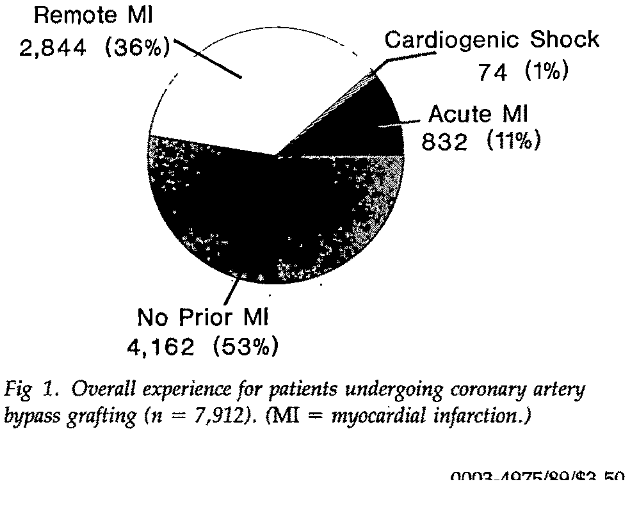

Fig 1. Overall experience for patients undergoing coronary artery bypass grafting (n = 7,912). (MI = myocardial infarction.)

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Table 1. Clinical Material: 1974–1987

No. of patients	832
Male sex (%)	77
Age (yr)	
Mean	61.4
Range	31–85
Total follow-up (patient-yr)	2,388
Mean (yr)	2.9
Completeness (1986–1987)	93%

Table 2. Clinical Presentation

Variable	No. of Patients	Percentage of Total
Angina	522	63
Diseased vessels		
1	59	8
2	135	20
≥3	441	64
Left main disease	52	8
Timing of operation		
≤24 h	132	16
1–7 days	302	36
8–14 days	225	27
15–30 days	173	21

The majority of patients, 63%, had postinfarction angina as a clinical indication for operation (Table 2). The patients without angina were operated on because of angiographic evidence of precarious coronary anatomy. Sixty-four percent of patients had three or more diseased vessels. Many angiograms were not available for review at the time of data entry into the computer, primarily because they had been returned to the referring cardiologist, and thus, details could not be confirmed. Therefore, when the patients are subdivided on the basis of the number of vessels involved, the number of patients does not equal the total number of patients operated on. Eight percent of the patients, 40% of whom did not have postinfarction angina, had clinically significant left main disease. More than half of the patients underwent operation within the first week after infarction.

Patient Management and Operative Technique

Preoperative intraaortic balloon pump support is used only for patients who are too unstable hemodynamically to undergo cardiac catheterization. Emergency CABG is performed for postinfarction angina patients who are refractory to intravenous nitroglycerin therapy. Limiting use of the intraaortic balloon pump avoids morbidity in patients who seldom need mechanical support after coronary revascularization. Our technique for CABG uses mild hypothermia (30°C) with intermittent aortic cross-clamping and fibrillation without cardioplegia. The first vessel to be grafted is the largest vessel, with the highest grade flow-limiting lesion without complete occlusion, that supplies the greatest portion of jeopardized but viable myocardium. Aortocoronary vein grafts are used routinely and, since 1983, the internal mammary artery increasingly is the preferred conduit.

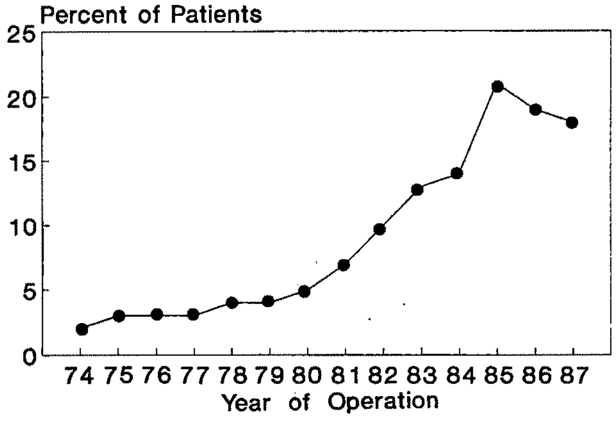

Fig 2. Distribution of patients with acute infarction.

Data Management

A prospective annual follow-up method is used in which experienced research assistants contact patients by telephone or questionnaire. Patients are followed on a continual basis, independent of any particular review for publication. All patient complications are confirmed by medical records, and causes of death are determined from autopsy reports, final summaries, or death certificates. Operative mortality included all deaths occurring within 30 days of operation and deaths occurring more than 30 days after operation if clearly related to the operation. Event-free curves were constructed according to the actuarial method [1]. Comparisons of event-free curves were made according to the method of Lee and Desu [2]. Univariate analysis of mortality was performed for the following factors: age, sex, postinfarction angina, extent of coronary artery disease (number of diseased vessels and left main disease), ejection fraction, left ventricular end-diastolic pressure, timing of operation, and number of bypass grafts. A Cox regression analysis [3] was performed for mortality using age, left ventricular end-diastolic pressure, and number of diseased vessels as continuous variables and sex, postinfarction angina, left main disease, and timing of operation as categorical variables.

Results

Operative Mortality

Operative mortality for patients undergoing CABG within 30 days after acute infarction is 4.7%. As shown in Table 3, the differences in operative mortality were not significant among the four timing groups. When the 132 patients operated on within 24 hours were compared with

Table 3. Operative Mortality

Variable	*p* Value	No. of Deaths	Operative Mortality (%)
Angina	0.04		
Yes		31	5.9
No		8	2.6
Diseased vessels	0.01		
1		0	0
2		1	0.7
≥3		22	5.0
Left main	. . .	5	9.6
Timing of operation	0.4		
≤24 h		10	7.6
1–7 days		12	4.0
8–14 days		9	4.0
15–30 days		8	4.6

the 700 patients operated on from day 2 through day 30, the difference in operative mortality was still not statistically significant ($p = 0.09$). Operative mortality is significantly increased by the extent of anatomical disease and the presence of postinfarction angina. Operative mortality decreased from 6.7% (7/104) before 1981 to 4.4% (32/728) since 1981 (Fig 3).

The causes of operative death are shown in Figure 4. Sepsis, aortic dissection, pneumonia, multiple-system failure, hemorrhagic cerebral vascular accident, and tamponade are included in the "other" category.

Hospital Stay

Length of hospital stay averaged 11 days for patients operated on within 24 hours of infarction compared with nine days for patients operated on after the first 24-hour period ($p = 0.02$).

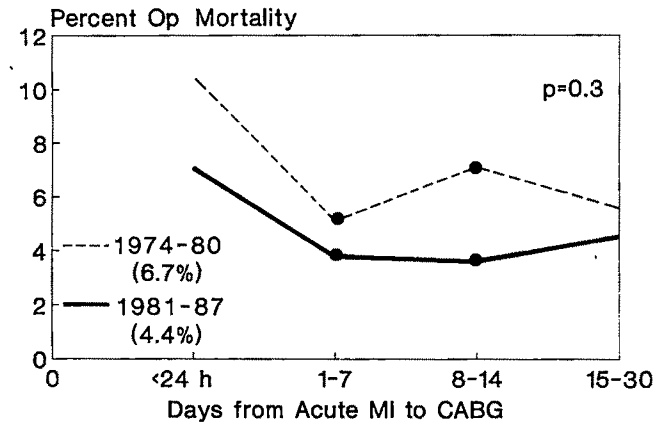

Fig 3. Operative mortality by time frame. (CABG = coronary artery bypass grafting; MI = myocardial infarction; OP = operative.)

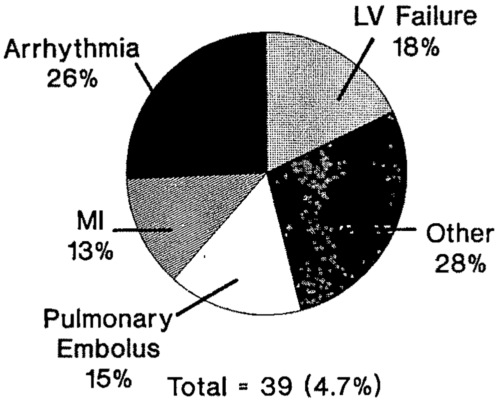

Fig 4. Causes of operative death. (LV = left ventricular; MI = myocardial infarction.)

Long-Term Survival

By univariate analysis, long-term survival was significantly affected by patient age (Fig 5) and left ventricular end-diastolic pressure (Fig 6). Presence or absence of postinfarction angina, patient sex, extent of coronary artery disease, ejection fraction, timing of operation, and number of bypass grafts were not significantly related to long-term survival.

The same two variables (age and left ventricular end-diastolic pressure) were significant according to the Cox multiple-regression analysis, and the other variables did not play a statistically significant role in survival when considered simultaneously (Table 4).

Comparison With Other Groups

Long-term survival of patients operated on after acute infarction was comparable with that of patients operated on after remote infarction (Fig 7). However, both of these infarction groups have a significant decrease in survival at 10 years compared with patients without previous infarction. The risk factors for these three groups are shown in Table 5. There were no statistically significant differences

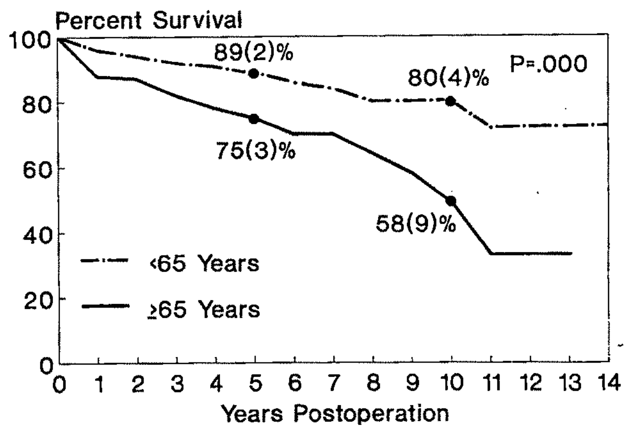

Fig 5. Survival by patient age.

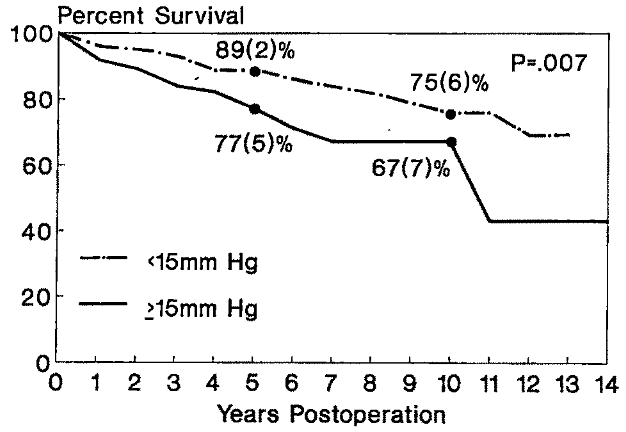

Fig 6. Survival by left ventricular end-diastolic pressure.

in freedom from postoperative myocardial infarction among the three groups (Fig 8).

Comment

Infarct Extension

After acute myocardial infarction, patients are at varying risk of recurrent infarction or infarct extension [4]. As a result, the overall mortality in the first year ranges between 10% and 15%, with more than one third of the deaths occurring in the first 6 weeks and one half occurring in the first 3 months after infarction [5, 6]. If postinfarction angina is present or results of a treadmill test are positive, the patients are at increased risk of infarct extension [7, 8]. This is consistent with the finding that 10% of patients recovering from acute infarction have critical narrowing of the left main coronary artery and 30% have clinically significant triple-vessel disease [9]. Another study showed that 54% of patients with acute anterior wall infarction and 25% of patients with inferior wall infarction had subtotal obstruction of the left anterior descending coronary artery [10].

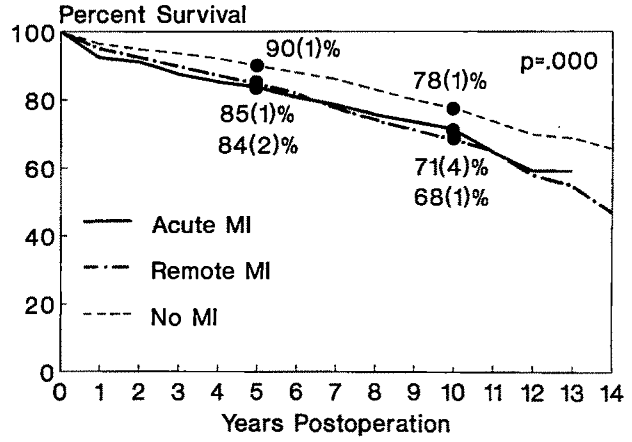

Fig 7. Long-term actuarial survival, comparing patients with acute myocardial infarction (MI) with those with remote infarction or no previous infarction.

In patients with postinfarction angina, recurrent infarction and infarct extension bear a high mortality rate, ranging between 15% for Q-wave infarctions and 43% for non–Q-wave infarctions [11]. A further study at the Mayo Clinic shows that of patients with subendocardial infarction 30% have subtotal occlusion of the left anterior descending coronary artery frequently associated with other clinically significant disease [12]. Extension occurs in 35% to 45% of anterior transmural infarctions, usually within hours, with a peak extent within seven to 14 days [13]. The incidence of in-hospital extension varies from 6% to 31%, and patients with extension have a mortality that is two to five times greater than that for primary infarction. The long-term prognosis is worse in patients with extension, with decreased survival at 1 year, especially in those with nontransmural infarction [14]. Rogers and co-workers [15] reported that 3-year survival in patients treated surgically (93%) was better than that of patients treated medically (64%) when residual jeopardized myocardium was present.

Operative Mortality

Controversy persists regarding the relative risks and appropriate timing of operative intervention during the postinfarction period. Some researchers suggest postponing surgical intervention. Roberts and associates [16] reported 20 patients with postinfarction angina managed medically for a mean of ten days before CABG with no operative deaths. Hochbert and colleagues [17] reported

Table 4. Results of Cox Regression Analysis of Long-Term Survival

Variable	p Value
Age	0.000
LVEDP	0.002
Extent of coronary disease	0.335
Sex	0.399
Timing of operation	0.650
Postinfarction angina	0.751

LVEDP = left ventricular end-diastolic pressure.

Table 5. Comparison of Risk Factors for the Infarction Groups

Variable	Acute MI	Remote MI	No MI	p Value
Diabetes	16%	13%	11%	<0.001
Hypertension	46%	40%	43%	0.001
Cigarette smoking	62%	56%	55%	<0.001
Family history	48%	41%	43%	0.004
Hyperlipidemia	21%	18%	18%	0.125
Cardiomegaly	32%	50%	44%	<0.001
Mean age (yr)	61.4	61.4	60.8	0.2
LVEDP (mm Hg)	29	26	17	<0.001

LVEDP, left ventricular end-diastolic pressure; MI = myocardial infarction.

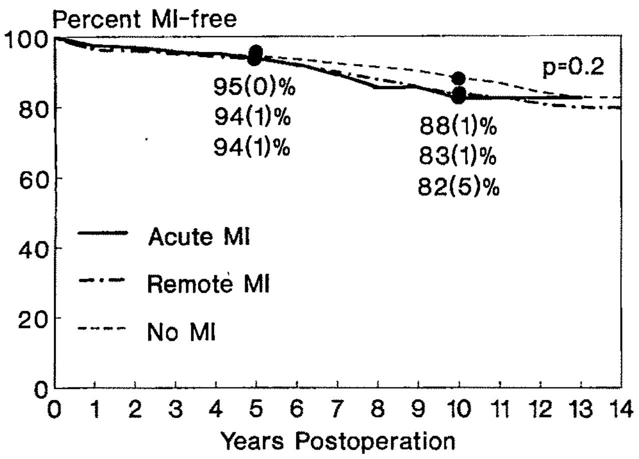

Fig 8. Freedom from postoperative myocardial infarction (MI).

that timing of CABG after infarction was not important if left ventricular function was normal but suggested that CABG should be delayed for 1 month after infarction if the ejection fraction was less than 50%.

Coronary artery bypass grafting has been performed in greater numbers of patients early after infarction to prevent further necrosis of jeopardized myocardium [18]. In the setting of postinfarction angina, some investigators report a mild increase in operative mortality [19, 20], whereas others report an operative mortality approaching that of elective CABG [21–23].

We showed that the presence of postinfarction angina and extent of disease are important predictors of operative mortality. Naunheim and co-workers [21] showed that three factors predict operative mortality: left ventricular function, clinical status, and age. The clinical status of the patients is the most important predictor of mortality, as operative mortality for postinfarction cardiogenic shock runs from 48% [21] to 63% [22]. The operative mortality for patients in cardiogenic shock in this study was 38%.

Long-Term Results

Although the presence of postinfarction angina does influence the operative mortality of CABG early after infarction, it does not appear to affect long-term survival. Similarly, the extent of disease affects operative mortality, but when considered separately and by the Cox regression model it was not an independent predictor of long-term survival. Timing of operation early after acute infarction had no significant effect on either operative mortality or long-term survival. For patients operated on within 30 days of infarction, long-term survival is primarily affected by the age and left ventricular function of the patients.

Conclusions

Operative mortality for patients undergoing CABG within 30 days after acute infarction is low and is independent of the time between infarction and operation. Operative mortality is significantly increased by the extent of anatomical disease and the presence of postinfarction angina.

Long-term survival for patients operated on within 30 days of infarction is affected by patient age and left ventricular function and is comparable with that for patients operated on after remote infarction. However, both of these preoperative infarction groups have a significant decrease in survival at 10 years compared with patients without previous infarction.

If ischemia or threatening coronary anatomy is present after acute infarction, early operation should be considered, as delaying CABG does not reduce the operative mortality and may increase the risk of recurrent infarction or infarct extension.

We appreciate the support of Janelle R. Wakefield and Joseph M. Grunkemeier in the preparation of the manuscript.

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DISCUSSION

DR RICHARD S. TOON (Des Moines, IA): At Mercy Hospital in Des Moines, IA, we tend to operate during the first hospital admission on patients who have sustained an acute myocardial infarct. This is a natural corollary to our work with emergency operations on evolving myocardial infarct. Once operation has been recommended, however, social concerns are the primary reason for a second admission for our patients.

This study by Floten and associates reports no difference in morbidity or mortality of early versus late operation; however, an early postdischarge mortality of up to 8% has been reported in patients sustaining acute myocardial infarct. In other words, this study has stacked the cards against early operation, because some of the patients destined for a second admission never make it to the catheterization laboratory because they die after their discharge, yet the results of their surgical groups are equal. If they were to compare the group of patients with acute myocardial infarct operated on during the first hospital admission with an equal group that would be catheterized, discharged, and scheduled for an operative procedure during a second admission, I believe that early operation would demonstrate superior results.

Dr Floten notes that 16% of the patients undergoing operation in the first 24 hours after myocardial infarction and 36% of the patients who were operated on during the first week had pain. How many of these patients can be classified as still having an acute evolving myocardial infarct or extension of their original infarct?

DR ANDREW C. FIORE (St. Louis, MO): I applaud Dr Floten and associates for their excellent operative results and their meticulous long-term follow-up. We support their conclusion that revascularization is safe after myocardial infarction.

At St. Louis University, 336 patients were operated on after sustaining an infarct. The overall mortality was 7.7%; this included patients operated on with preoperative use of an intraaortic balloon pump and those in cardiogenic shock. By multivariate stepwise logistic regression analysis, we determined that the predictors of mortality were age, preoperative left ventricular function, and the clinical status of the patient at the time of operation. The time interval from infarct to bypass grafting was not a significant independent predictor of mortality.

We agree with Floten and associates that there is no advantage in delaying myocardial revascularization after infarction. However, when operation does occur, it is the preoperative clinical status of the patient and the left ventricular function that determine the operative risk. I have one question. The analysis of long-term survivors showed age to be an independent risk factor of operative mortality. Dr Floten, could you comment on the left ventricular function of this patient population? We believe that it is a significant predictor of operative mortality.

DR CARY W. AKINS (Boston, MA): It would be of interest if Floten and associates could distinguish between subendocardial and transmural myocardial infarction in their study. Although the hospital mortality rate for transmural infarction is greater than that for subendocardial infarction, in long-term follow-up, certainly subendocardial infarction is associated with poorer longevity. Maybe that was an indication for operation, but more than 30% of the patients did not have angina. What was their indication for operation? Although Floyd Loop has said that myocardial infarction is a good indicator of coronary artery disease, I do not believe that we can make the extrapolation that it is an indicator for operation. It would also be of interest to know how many patients with angina had ischemia at a distance from the infarction or in the same zone.

DR ROBERT A. GUYTON (Atlanta, GA): I applaud Floten and associates on their follow-up, which is a difficult achievement. I wish to know if their data are sufficient to tell us whether one should delay operation one day, two days, three days, or four days after an infarction occurs. Can this question be answered for a patient who is relatively stable after a large infarction?

We also have the clinical impression that a right ventricular infarction is different from a left ventricular infarction. If a patient has a large, clinically significant right ventricular infarction there appears to be a considerable advantage to waiting a week or 2 weeks after infarct. Dr Floten, do you have an opinion on this point?

DR LAWRENCE L. MICHAELIS (Chicago, IL): My associates at Northwestern and I recently analyzed the same type of patients. There was a very significant increase in mortality in patients who had a subendocardial infarction compared with those who had a transmural infarction. This is somewhat surprising to us, but we believe that the possibility of reperfusion injury during early operation is much greater in patients who have had a subendocardial infarction rather than a completed transmural infarction.

Dr Floten, have you differentiated between these two types of infarction? Several other recent articles have corroborated our data, so I think it is of extreme importance when recommending early operation to this group of patients.

DR FLOTEN: I will try to respond to the comments and questions. Dr Toon, we have not addressed the issue of the numbers of patients in the first week who undergo recurrent infarction or extension of infarction. We know from the medical literature that the mortality rate is about 45% for patients who have a recurrent or extended infarction with concurrent non–Q-wave or subendocardial infarction. In our series we have not distinguished between the patients who were having a true recurrent infarction four or five days after the original event and those with a continuing, evolving infarction.

Dr Fiore brings out a good point—we did not evaluate left ventricular function in the early patient groups that were operated on within 24 hours or in the first week. However, patients operated on within the first 24 hours usually were a sicker, more unstable group of patients because almost all of them were receiving nitroglycerin or dopamine, indicating an evolving infarction.

The issue of ischemia at a distance from the original infarction touches on what I have just said. Ischemia at a distance or a recurrent infarct in the face of a subendocardial myocardial infarction has a much higher mortality rate than transmural myocardial infarction that is continuing as a localized infarction.

We did not address the issue of reperfusion injury for subendocardial infarction versus transmural infarction. Many previous investigators have examined the predictors of operative mortality and some of the other issues. The basis of our study has been to strengthen the case for earlier operation after infarction by showing that long-term results are not improved by a delay in operation.

Elevated Hemidiaphragm After Cardiac Operations: Incidence, Prognosis, and Relationship to the Use of Topical Ice Slush

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Richard A. Schmaltz, MD, Theresa Boley, BSN, Richard Madsen, PhD,
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We have reviewed chest roentgenograms of 745 patients before hospital dismissal after cardiac operations and serially to determine the incidence and prognosis of elevated hemidiaphragm and any relationship to the use of topical ice slush (TIS) in myocardial preservation. All patients had similar myocardial preservation techniques including moderate systemic hypothermia and 4°C saline solution poured over the heart at aortic clamping. During a 12-month period, TIS was added to the saline bath. Two (2.4%) of 84 patients before TIS and 5 (2.5%) of 201 consecutive patients operated on since discontinuing TIS had elevated hemidiaphragm on the predismissal roentgenogram. Of 460 patients in whom TIS was employed, 109 (23.7%) had elevated hemidiaphragm ($p < 0.001$). When TIS was employed, elevated hemidiaphragm developed in 72 (26%) of 280 patients without internal mammary artery takedown versus 13 of 33 patients (39.4%) with takedown of the internal mammary artery ($p = 0.047$). Ninety-nine patients with elevated hemidiaphragm were available for follow-up at 1 month, at which time 79 (79.8%) continued to have elevated diaphragm. At 1 year, 14 (21.9%) of 64 patients had persistent diaphragm elevation. We conclude that TIS predisposes to elevated diaphragm and that the incidence is increased when the internal mammary artery is harvested.

(Ann Thorac Surg 1989;48:764–8)

Although a variety of myocardial preservation techniques are used in open heart surgery today, the key principle with them is decreasing myocardial oxygen consumption by maintaining a cold myocardium. For several years, we have applied cold saline solution topically at the time of aortic cross-clamping to aid in cooling the myocardium. Topical ice slush (TIS) was then added to this regimen as a further means of reducing myocardial temperature rapidly. It became apparent that we were observing an increased incidence of elevated hemidiaphragm in our patients postoperatively, particularly in patients who had undergone internal mammary artery harvesting. This report details our retrospective and prospective studies that were performed to determine the incidence and prognosis of elevated hemidiaphragm after cardiac operations and any relationship to the use of TIS or harvesting of the internal mammary artery. Additionally, we wished to determine any morbidity or mortality associated with elevation of hemidiaphragm.

Material and Methods

During the last 9 years at the University of Missouri Hospital and Clinics, myocardial preservation in adults has been accomplished by injecting 1 L of 4°C crystalloid cardioplegia directly into the aortic root at the time of aortic clamping with reinfusion of 200 mL of cardioplegia every ten to 15 minutes during the period of cross-clamping. Other measures used to insure that the myocardium is maintained cold include cooling the patient systemically to 22° to 25°C and the application of 1 L of 4°C saline solution poured rapidly over the heart at the time of initial aortic clamping. An additional liter of 4°C saline solution is drained into the pericardium during the two to four minutes required for the infusion of the initial bolus of cardioplegia, with excess saline solution being aspirated from the pericardium. In 1983, in an effort to more rapidly cool the myocardium, we began applying 1 L of ice slush of "melting snow" consistency at the time of aortic clamping in lieu of the initial liter of saline solution. Because of the known risk of cold injury to the heart or surrounding structures, care was taken to avoid any particles of ice larger than those of "snowcone" consistency. In general, the TIS turned to water during the pericardial infusion of the liter of 4°C saline solution and almost always was absent from the pericardium within ten minutes. After suspecting that the TIS may have been responsible for an observation of bilateral phrenic nerve paralysis, we eliminated the slush, returning to the original practice of using 4°C saline solution.

Chest roentgenograms on 84 cardiac surgery patients operated on before the institution of TIS were available for review. These patients are referred to as group 1. Four hundred sixty consecutive patients with acquired cardiac

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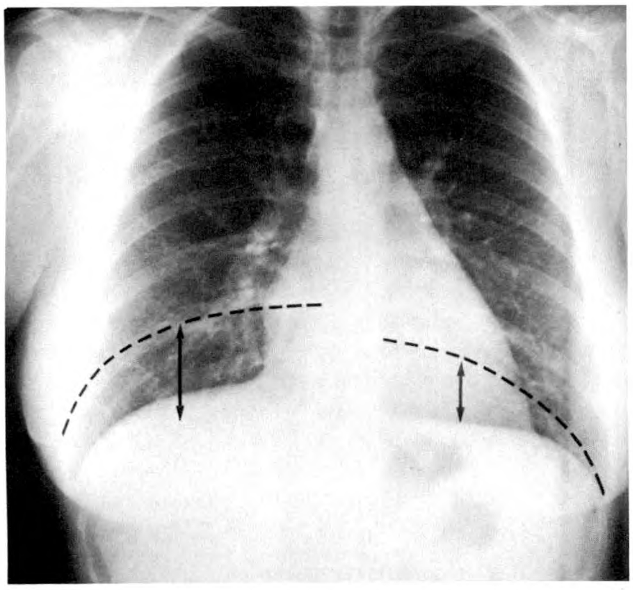

Fig 1. Criteria for elevated hemidiaphragm. To be considered elevated, the right hemidiaphragm had to be greater than two rib spaces higher than the left hemidiaphragm. For the left hemidiaphragm to be considered elevated, it had to be greater than one rib space higher than the right hemidiaphragm.

disease were operated during the period when TIS was employed and are referred to as group 2. After TIS was discontinued, x-ray films were reviewed on 201 consecutive cardiac surgery patients; these patients are referred to as group 3.

Predismissal chest x-ray films were reviewed on these 745 patients and compared with preoperative films for evidence of elevated diaphragm. Roentgenograms were then reviewed at 1 month postoperative and serially in patients with elevated diaphragm to document recovery. The arbitrary criteria for considering a diaphragm elevated are shown in Figure 1. Because the right hemidiaphragm normally is slightly higher than the left, to consider the right hemidiaphragm elevated, it had to be greater than two intercostal spaces higher than the left hemidiaphragm. For the left hemidiaphragm to be elevated, it had to be greater than one intercostal space higher than the right hemidiaphragm. All x-ray films were reviewed by one individual (W.N.) to avoid interobserver variability.

The groups were compared using the χ^2 test for categorical variables. Continuous variables between groups were analyzed with Wilcoxon rank sum tests.

Results

More than 3,000 chest x-ray films were reviewed for 745 patients. The significant findings of this study are summarized in Table 1. During the period 1982 to 1983, before TIS was used, 2 (2.4%) of 84 patients had elevated hemidiaphragm. During the period of employing topical slush, beginning in 1983, 109 (23.7%) of 460 consecutive adult patients had elevated hemidiaphragm. After TIS was discontinued in early 1985, 5 (2.5%) of 201 consecutive patients had elevated hemidiaphragm. The increased incidence of elevated hemidiaphragm with the use of TIS is highly significant ($p < 0.001$).

Table 1. Observed Proportion of Postoperative Elevated Hemidiaphragm

Group	No. of Patients	Elevated Hemidiaphragm No.	Percent[a]
1 (before TIS)	84	2	2.4
2 (TIS employed)	460	109	23.7
3 (after TIS)	201	5	2.5

[a] $p < 0.001$ by χ^2 analysis.

TIS = topical ice slush.

Overall, nearly 95% of the observed diaphragm elevations were on the left side. The right hemidiaphragm was elevated in 5 patients (4.23%), and bilateral diaphragm involvement was present in 1 patient. There were no cases of a right hemidiaphragm elevation or bilateral diaphragm involvement in patients who did not have TIS used as a myocardial preservation adjunct.

Predictors of Elevated Diaphragm

Group 2 patients in whom TIS had been used were analyzed in further detail to determine risk factors that might result in elevated hemidiaphragm. Gender, patient's age, and aortic cross-clamp time were not significantly related to the observation of elevated hemidiaphragm (p values = 0.109, 0.838, and 0.094, respectively). There was, however, a difference in the incidence of elevated hemidiaphragm depending on the operation performed. Patients undergoing valvular operations had an observed incidence of elevated hemidiaphragm of 10.6% (7 of 66 patients). This contrast with a 27.2% (85 of 313 patients) incidence of elevated hemidiaphragm observed in patients undergoing isolated coronary artery bypass grafting and 20.9% (17 of 81 patients) in those undergoing complex cardiac procedures that also included coronary artery bypass grafting ($p = 0.013$).

Effect of Internal Mammary Artery Harvesting on Hemidiaphragm Elevation

Patients in group 1 were operated on before the revival of internal mammary artery bypass at this institution. Thirty-three of 313 patients in group 2 and 98 of 155 patients in group 3 undergoing isolated coronary artery bypass grafting did have internal mammary artery used as a conduit. Table 2 shows the effect of harvesting internal mammary artery on the incidence of elevated diaphragm during the period when ice slush was used and subsequently when this practice was discontinued. When TIS was employed, harvesting the internal mammary artery significantly increased the incidence of observed elevated hemidiaphragm ($p = 0.047$). When ice slush was not employed, no significant difference was observed in the rate of elevated hemidiaphragm whether or not the internal mammary artery was harvested ($p = 0.311$).

Table 2. Influence of Topical Ice Slush and Use of Internal Mammary Artery on Elevated Hemidiaphragm

Operation	Group 2 (TIS)	Group 3 (no TIS)
CABG with IMA	13/33 (39.4%)	3/98 (3.06%)
CABG without IMA	72/280 (25.7%)[a]	1/57 (1.75%)[b]

[a] $p = 0.047$ versus with IMA. [b] $p = 0.311$ versus with IMA.

CABG = coronary artery bypass grafting; IMA = internal mammary artery; TIS = topical ice slush.

Benefit of Topical Ice Slush

The overall hospital mortality was not different when considering all patients in groups 1, 2, and 3 (5.9%, 3%, and 3.9%, respectively). If one compares the hospital mortality in patients with isolated coronary artery bypass grafting in groups 1 and 3 who did not have ice slush employed with patients in group 2 who did have ice slush employed, a difference in hospital mortality is suggested, 4.1% versus 1.3% ($p = 0.037$). However, at a 95% confidence interval, the difference in hospital mortality between these groups could range from 0.11% to 5%.

Mortality and Morbidity Associated With Elevated Diaphragm

Only one death in this series was considered directly attributable to elevated diaphragm. This was the 1 patient who developed bilateral diaphragmatic paralysis and died after prolonged mechanical ventilation.

The hospital mortality in patients observed to have elevated diaphragm in group 2 was 1.8% (2 of 109 patients). This was not significantly different from the 3.4% mortality (12 of 351 patients) observed in patients in

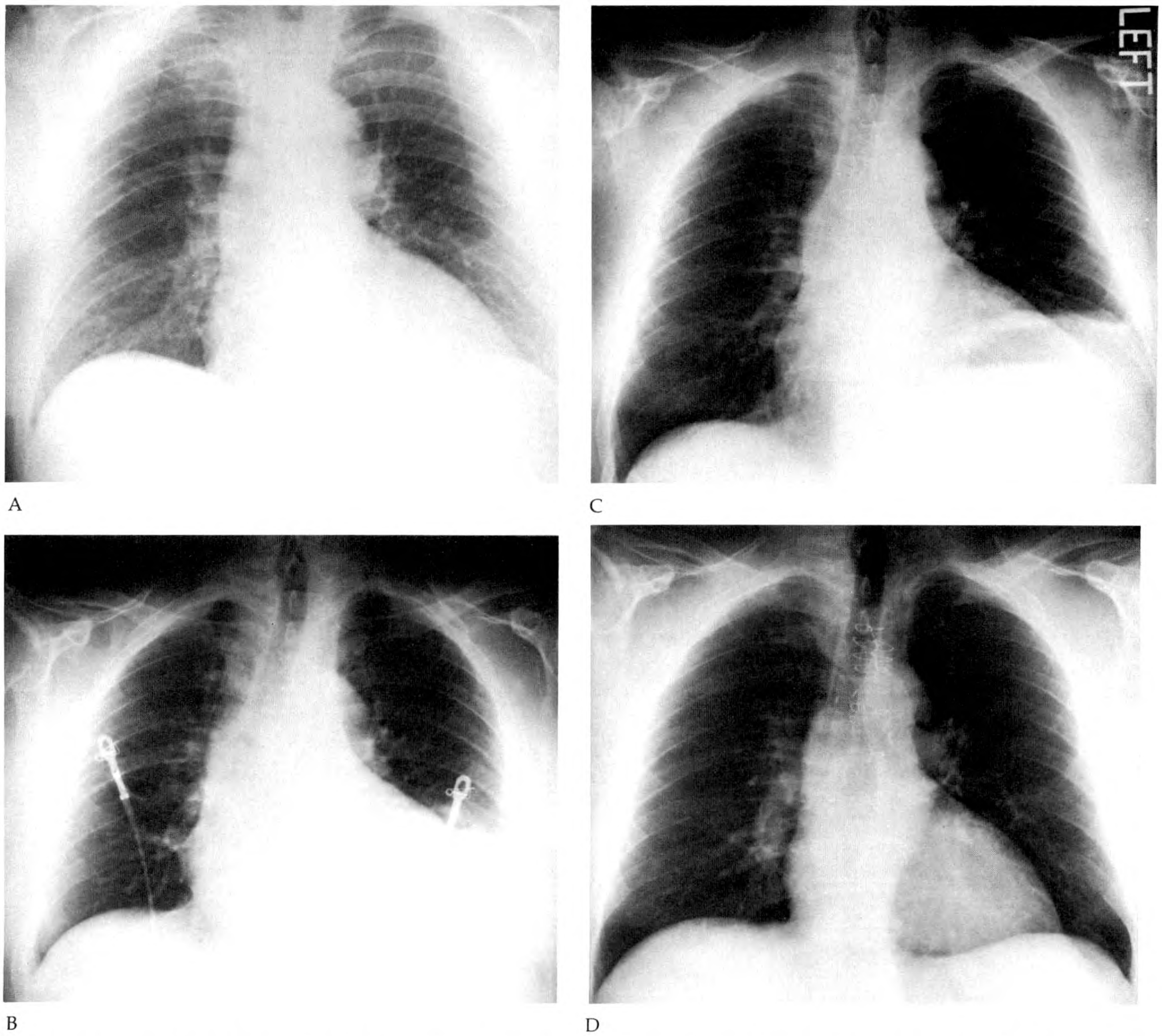

Fig 2. (A) Preoperative chest roentgenogram with normal diaphragm position. (B) Predismissal chest roentgenogram showing elevated left hemidiaphragm. (C) Left hemidiaphragm remains elevated at 1 month postoperative. (D) Left hemidiaphragm in normal position at 1 year after operation.

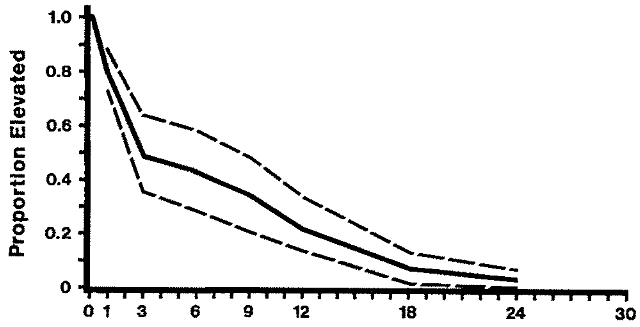

Fig 3. Point-wise persistent hemidiaphragm elevation at varying time intervals after open heart operation. Beginning with 109 patients with elevated hemidiaphragm, of the patients available for review, 79.8%, 21.9%, and 2.6% had persistent elevation of hemidiaphragm at 1 month, 1 year, and 2 years, respectively. The two interrupted lines *bracket a 95% confidence interval.*

whom ice slush was employed who did not have elevated diaphragm ($p = 0.168$).

The median duration of endotracheal intubation was not significantly different in patients with or without elevated hemidiaphragm (26.8 and 26.4 hours, respectively; $p = 0.352$). Also, the average hospital stay was identical in both groups (nine days; $p = 0.589$).

Recovery of Diaphragm Elevation

Figure 2 shows an example of recovery of hemidiaphragm to normal position. Of 99 patients available for follow-up at 1 month, 20 (20.2%) had return of a previously elevated hemidiaphragm to normal position. At 1 year, 50 (78.1%) of 64 patients had recovery of elevated hemidiaphragm, and at 2 years 74 (97.4%) of 76 patients available for study had normal diaphragm position.

Figure 3 shows a point-wise graph of persistent diaphragm elevation in these 109 patients plotted against time bracketed by 95% confidence interval.

Comment

Although the optimal method of myocardial preservation is far from settled, the importance of myocardial cooling to lower myocardial oxygen demand during cardiac operations is accepted. Cross and associates [1] reported that localized cardiac hypothermia accomplished by perfusing coronary arteries with cold blood after induction of potassium arrest could effectively extend the period of safe cardiac anoxia in animals. In 1959, Shumway and associates [2] described a technique of more profound selective hypothermia of the heart during cardiac arrest accomplished by continuous irrigation of the surface of the heart with normal saline solution at 4°C, and excellent early clinical results were obtained with this technique [3, 4]. In 1962 Speicher and co-workers [5] showed in both humans and dogs that myocardium and pericardium could be injured by ice slush directly applied to the heart and pericardium and suggested that continuous exposure to ice slush in the pericardium not exceed one hour. Scannell [6] called attention to the possibility of phrenic nerve injury secondary to local myocardial cooling with crushed ice and saline and coined the term "frost bitten phrenic." Marco and colleagues [7] subsequently presented experimental documentation in dogs that direct or indirect contact of the phrenic nerve with crushed ice for 30 or 60 minutes could result in phrenic paralysis. In their study, microscopical examination of injured phrenic nerves revealed demyelination and preserved axons with some fragmentation. This should not have been surprising as Webster had shown that peripheral nerve fibers are easily damaged by exposure to cold and may well be the most cold-sensitive mammalian tissue [8].

There have since been several clinical reports [9–15] of unilateral and bilateral phrenic nerve damage with the use of intense local cardiac cooling using crushed ice. However, the majority of these reports involved multiple applications and prolonged exposure of the pericardium to crushed ice. During our experience of using TIS for myocardial preservation, great care was taken to avoid large ice particles within the pericardial cavity, preferring an ice slush similar in consistency to melting snow. The slush was only occasionally applied more than once and was usually absent from the pericardial cavity to gross inspection within ten minutes of its application.

In this review, the definition of elevated diaphragm was arbitrary and is not intended to be synonymous with phrenic nerve paralysis. However, the incidence of hemidiaphragm elevation with use of ice slush (23.7%) compared with the 2.5% elevated diaphragm incidence on discontinuing TIS causes one to suspect some degree of cold-induced phrenic nerve injury. In a small number of patients tested by fluoroscopy or phrenic nerve conduction times, suspected pathology was always confirmed. The incidence of hemidiaphragm elevation reported in this series is consistent with the 24% incidence observed in the series by Rousou and associates [14].

In this series, the clinical recognition of an increased incidence of elevated hemidiaphragm was coincident with a revival of the use of internal mammary artery for coronary artery bypass grafting. Because the application of ice slush to the pericardium was brief and care was taken to avoid large particles of ice, we originally suspected that the increased incidence of hemidiaphragm elevation might be due to phrenic nerve injury secondary to internal mammary artery harvesting. This prompted us to continue these observations prospectively with group 3 patients in whom TIS was not employed but in whom use of the internal mammary artery as a conduit increased to 63% of all patients undergoing isolated coronary artery bypass grafting from the 10.5% rate in group 2 patients. In spite of the increased use of internal mammary artery, the incidence of elevated hemidiaphragm dropped from 27.2% to 2.5%. In patients who did not have TIS employed, harvesting the internal mammary artery was not a risk factor for the observance of elevated hemidiaphragm (3.06% when internal mammary artery was harvested versus 1.75% without internal mammary artery; $p = 0.311$). However, in patients who had TIS employed, harvesting the internal mammary artery did appear to cause an increase in the incidence of elevated diaphragm observed (39.4% versus 25.7%; $p = 0.047$).

The sobering realization that bilateral phrenic nerve paralysis was responsible for a patient's death prompted this review. Although several reports [6, 9, 15] of patients surviving bilateral diaphragm paralysis are available, this condition is poorly tolerated. This is easily understood as the diaphragm is responsible for 30% to 60% of the tidal volume in adults in the supine position [16]. Even unilateral hemidiaphragm paralysis can be catastrophic in young children, though it is generally well tolerated in adults [17, 18]. Abnormal pulmonary function tests have been demonstrated by Clague and Hall [19] in patients with unilateral hemidiaphragmatic paralysis. Our clinical impression has been that patients with elevated hemidiaphragm do less well, though this could not be demonstrated statistically in this study. Specifically, the duration of mechanical ventilation and hospital stay after a cardiac operation were not significantly different in patients in whom elevated hemidiaphragm did or did not develop. Also, hospital mortality was not significantly different in patients with elevated hemidiaphragm when compared with patients without elevated hemidiaphragm (1.8% versus 3.4%).

Recovery of cold-induced phrenic nerve injury was demonstrated by Marco and co-workers [7] in animals and has been documented in several clinical studies since. The time for recovery of diaphragmatic function has varied from 30 days to 2 years [7, 12, 13, 15]. This variation and the results of this series would indicate that the cold-induced injury is extremely variable. Although over 3,000 x-ray films were inspected in this series, timing of diaphragmatic recovery remains an estimate as x-ray films were obtained infrequently in any given patient. From a more careful follow-up in the 109 patients with elevated hemidiaphragm, recovery to normal position occurred in approximately 20% by 1 month, 78% by 1 year, and 97% by 2 years. Dajee and associates [12] reported a 75% recovery rate at 1 year.

Because of the recognized opportunity of topically applied ice to injure the phrenic nerve, a variety of techniques and barriers have been developed to avoid this complication, some with encouraging results [14, 20]. Interestingly, however, some of the cooling pads designed to cool the myocardium while avoiding use of ice have resulted in phrenic nerve damage as well [14].

We conclude that the use of topical ice slush as a local myocardial coolant, even for a brief time, is associated with a significant increase in the incidence of elevated hemidiaphragm. The propensity to elevated hemidiaphragm is increased further when the internal mammary artery is harvested, presumably because of an increased proximity of TIS to the phrenic nerve. Our clinical bias is that patients with elevated hemidiaphragm do less well than those without this finding even though recovery of diaphragm position is nearly complete at 2 years. Any possible benefit of TIS used as an adjunct for myocardial cooling must be evaluated with the knowledge of the potential risks associated with this technique.

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Cerebral Vasoreactivity to Carbon Dioxide During Cardiopulmonary Perfusion at Normothermia and Hypothermia

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With the pH-stat acid-base regulation strategy during hypothermic cardiopulmonary bypass (CPB), carbon dioxide (CO_2) is generally administered to maintain the partial pressure of arterial CO_2 at a higher level than with the alpha-stat method. With preserved CO_2 vasoreactivity during CPB, this induction of "respiratory acidosis" can lead to a much higher cerebral blood flow level than is motivated metabolically. To evaluate CO_2 vasoreactivity, cerebral blood flow was measured using a xenon 133 washout technique before, during, and after CPB at different CO_2 levels in patients who were undergoing coronary artery bypass grafting with perfusion at either hypothermia or normothermia. The overall CO_2 reactivity was 1.2 mL/100 g/min/mm Hg. There was no difference between the groups. The CO_2 reactivity was not affected by temperature or CPB. The induced hemodilution resulted in higher cerebral blood flow levels during CPB, although this was counteracted by the temperature-dependent decrease in the hypothermia group. After CPB, a transient increase in cerebral blood flow was noted in the hypothermia group, the reason for which remains unclear. The study shows that manipulation of the CO_2 level at different temperatures results in similar changes in cerebral blood flow irrespective of the estimated metabolic demand. This finding further elucidates the question of whether alpha-stat or pH-stat is the most physiological way to regulate the acid-base balance during hypothermic CPB.

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The interpretation and application of optimal acid-base management during hypothermic cardiopulmonary bypass (CPB) is a matter of controversy. Two different strategies, pH-stat and alpha-stat management, are employed [1, 2]. Alpha-stat management implies that a *nontemperature-corrected* partial pressure of arterial carbon dioxide ($PaCO_2$) of about 40 mm Hg is maintained during the entire CPB period. The rationale is to maintain a constant buffer capacity of alpha-histidine-imidazole [1]. In contrast, pH-stat management implies that the *temperature-corrected* $PaCO_2$ is kept at about 40 mm Hg to maintain a pH close to 7.42 at the actual temperature. To maintain pH-stat during hypothermic CPB, it is generally necessary to increase the CO_2 content of the gas inflow to the perfusate. Consequently, during hypothermic CPB when pH-stat acid-base management is selected, the increased administration of CO_2 results in respiratory acidosis according to the alpha-stat theory.

At present, most cardiopulmonary perfusions are performed at moderate hypothermia, mainly for reasons of myocardial protection, although the reduced metabolic demand and blood flow are also beneficial for the brain. A lower cerebral blood flow (CBF) is expected to decrease the risk of cerebral microembolism [3].

Carbon dioxide is known to be a potent cerebral vasodilator at normal body temperature [4]. If this is true also at hypothermia, the increased administration of CO_2 during pH-stat management leads to vasodilatation that endangers cerebral autoregulation [5] and increases CBF, which may counteract the beneficial effect of hypothermia.

In a previous report, we [6] demonstrated that CBF during hypothermic CPB remained unchanged from the level measured before CPB when alpha-stat acid-base management was employed. There was no evidence of cerebral hyperemia except for a short-lived increase in CBF after termination of CPB.

To gain further support for the alpha-stat acid-base mode, the cerebral vasoreactivity to changes in $PaCO_2$ was determined during normothermic and hypothermic CPB. The oxygen delivery rate to the brain ($CDRo_2$) was also calculated to estimate whether the pattern of changes in CBF and $CDRo_2$ during and after CPB indicates that different CBF levels during normothermic and hypothermic CPB affect the risk for brain damage.

Material and Methods

Eighteen male patients participated in the study. They were randomly selected from those admitted to the Department of Thoracic Surgery, University Hospital, Lund, for coronary artery bypass grafting. Only individuals without signs or symptoms of cerebrovascular or carotid artery disorders were chosen. The study was approved by the local ethics committee at the University of Lund.

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Table 1. Characteristics of the Two Patient Groups[a]

Variable	Hypothermia Group (n = 9)	Normothermia Group (n = 9)	p Value[b]
Age (yr)	56 ± 9	57 ± 7	NS
Weight (kg)	86 ± 15	85 ± 10	NS
Height (cm)	177 ± 8	172 ± 6	NS
Body surface area (m^2)	2.0 ± 0.1	2.0 ± 0.1	NS
Perfusion time (min)	116 ± 13	75 ± 21	$p < 0.001$
No. of bypass grafts	3.9 ± 0.8	3.2 ± 0.8	$p < 0.05$
Total fentanyl (mg)	7.6 ± 2.0	6.1 ± 1.6	NS
Total droperidol (mg)	18.5 ± 7.3	16.3 ± 5.6	NS
Anesthesia time (min)	384 ± 47	348 ± 47	NS

[a] Data are shown as the mean ± the standard deviation. [b] Values are from the unpaired t test.

NS = not significant.

Informed consent was obtained from each patient before the investigation.

The patients were divided into two equal groups (Table 1). In 9 patients, CPB was performed during moderate hypothermia (approximately 30°C), whereas the other 9 were perfused at the existing body temperature (approximately 35°C).

Cardiopulmonary Bypass

Cardiopulmonary bypass was performed in the nonpulsatile mode using a Stöchert roller pump and a Cobe CML membrane oxygenator. A 40-μm Bentley arterial catheter filter was used. The tubing consisted of silicon in the pump head and polyvinyl chloride in the catheter. The system was primed with 2,000 mL of Ringer's lactate solution, 250 mL of 15% mannitol, and 75 mmol/L of Addex tromethamine. The coronary suckers were connected to a Bentley cardiotomy reservoir (model 3538), which includes a 20-μm filter. The pump flow was initially held at about 2.5 L/min/m^2 followed by a small reduction during hypothermia to maintain mean arterial blood pressure of greater than 50 mm Hg during the entire period of perfusion.

In the hypothermia group, chlorpromazin hydrochloride (5 to 10 mg) was used in 4 patients to reduce blood pressure and to enhance rewarming at the end of CPB. One other patient in this group needed inotropic support (dopamine hydrochloride) to increase blood pressure after the induction of anesthesia and also after CPB. None of the remaining 4 patients in this group and no patients in the normothermia group were treated with any drugs to keep blood pressure within acceptable limits (50 to 90 mm Hg during CPB). Carbon dioxide was added to the oxygenator to regulate the $PaCO_2$ level. The $PaCO_2$ was kept regulated according to alpha-stat perfusion.

Cerebral Blood Flow Measurements

Regional hemispheric CBF was measured after intraarterial administration of 0.1 GBq (2 mCi) of xenon 133. The tracer substance was injected through a catheter positioned under direct vision in the right common carotid artery, and clearance of the tracer was monitored by five external scintillation detectors positioned over the right parietotemporal region. The Novo cerebrograph model 10a was used for data collection and subsequent calculation of CBF by the method of Olesen and co-workers [7]. Thus, the CBF values correspond mainly to the gray matter flow. All CBF values are presented as the hemisphere mean. Corrections for changing xenon solubility in blood caused by variation in hemoglobin concentration were performed [8].

Operative Data

Droperidol (2.5 to 5 mg) and fentanyl (0.05 to 0.1 mg) were given as premedication. Anesthesia was induced and maintained as described in a previous report [6]. Total doses of droperidol and fentanyl along with other group characteristics are shown in Table 1. All patients were ventilated with a gas mixture of 50% nitrous oxide in O_2 (before and after CPB). In both groups, the ventilation was adjusted to maintain the $PaCO_2$ within normocapnic levels analyzed at 37°C.

In all patients, three paired CBF measurements were performed during operation, as shown in Figure 1. The first pair and the third pair were done before and after CPB, respectively, and the second was done during hypothermic or normothermic CPB. The cerebrovascular response to CO_2 was evaluated by altering the $PaCO_2$ in the second CBF measurement in each pair about 25 minutes after each control measurement during steady-state conditions. In each group, the alteration of $PaCO_2$ within each pair of measurements was performed randomly from a higher to a lower level in 5 patients and in reverse order in the other 4. This was achieved by

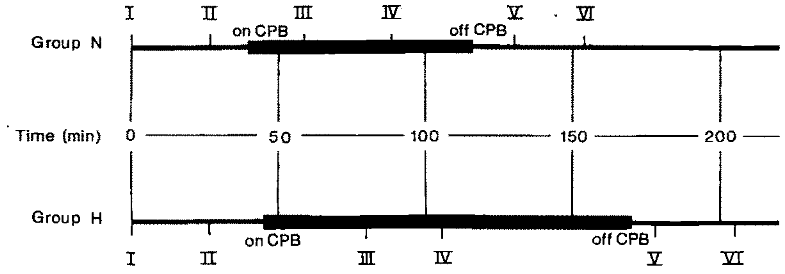

Fig 1. Relationship between paired measurements of cerebral blood flow (roman numerals) *and cardiopulmonary bypass* (CPB) *for the two groups. All measurements were conducted during operation.* (H = *hypothermia;* N = *normothermia.)*

Table 2. Cerebral Blood Flow and Related Data Under Conditions of Higher and Lower Partial Pressure of Arterial Carbon Dioxide for the Two Groups[a,b]

		Before CPB		During CPB		After CPB	
Variable	Group	Higher	Lower	Higher	Lower	Higher	Lower
CBF (mL/100 g/min)	H	34 ± 14	25 ± 15^c	40 ± 10	22 ± 7^c	43 ± 16	25 ± 6^c
	N	32 ± 10	19 ± 5^c	46 ± 8	28 ± 5^c	46 ± 14	27 ± 6^c
External auditory temperature (°C)	H	34.9 ± 0.5	34.9 ± 0.5	28.3 ± 1.1^d	29.2 ± 1.0^d	36.2 ± 1.0^d	36.1 ± 0.9^d
	N	34.5 ± 0.5	34.5 ± 0.5	34.6 ± 0.7	34.7 ± 0.6	34.6 ± 0.6	34.7 ± 0.5
$PaCO_2$ (mm Hg)	H	45 ± 3	33 ± 3^c	48 ± 3	33 ± 2^c	42 ± 5	31 ± 5^c
	N	45 ± 4	33 ± 2^c	44 ± 3	31 ± 4^c	42 ± 5	30 ± 3^c
Arterial blood pressure (mm Hg)	H	72 ± 11	73 ± 15	73 ± 18	62 ± 22	70 ± 6	66 ± 15
	N	74 ± 13	68 ± 11	75 ± 13	67 ± 16	67 ± 12	65 ± 8
Hemoglobin (g/L)	H	143 ± 15	142 ± 13	103 ± 15	104 ± 13	113 ± 14	116 ± 16
	N	139 ± 12	139 ± 10	98 ± 15	98 ± 9	104 ± 10	106 ± 10
pH (U)	H	7.374 ± 0.03	7.456 ± 0.03^c	7.341 ± 0.04	7.460 ± 0.04^c	7.367 ± 0.04	7.467 ± 0.06^c
	N	7.344 ± 0.02	7.456 ± 0.03^c	7.365 ± 0.03	7.488 ± 0.06^c	7.349 ± 0.05	7.447 ± 0.06^c
PaO_2 (mm Hg)	H	131 ± 27	122 ± 30	149 ± 45^d	136 ± 38	137 ± 75	128 ± 67
	N	134 ± 32	143 ± 30	109 ± 29	111 ± 29	124 ± 43	132 ± 43

[a] For all measurements, there were 9 patients in each group. The data in the column "Higher" before CPB consist of values from the first measurement in 5 patients and from the second measurement in 4, and so on. See text for details. [b] Data are shown as the mean ± the standard deviation. [c] Significance: $p < 0.05$ between higher and lower values. [d] Significance: $p < 0.05$ between the two groups.

CBF = cerebral blood flow; CPB = cardiopulmonary bypass; H = hypothermia; N = normothermia; $PaCO_2$ = partial pressure of arterial carbon dioxide; PaO_2 = partial pressure of arterial oxygen.

changing the ventilation (before and after CPB) or the amount of CO_2 added to the oxygenator (during CPB).

Arterial blood pressure, measured in the left radial artery, and central venous pressure, measured in the superior vena cava, were continuously monitored and recorded. The esophageal, arterial-in-line, and external auditory meatus temperatures were measured repeatedly. Arterial blood, sampled immediately after injection of the tracer, was analyzed for arterial blood gases, pH, and hemoglobin at 37°C. The results were not corrected for deviations in body temperature from 37°C.

Cerebral Delivery Rate of Oxygen

The $CDRo_2$ was calculated from the following formula:

$$CDRo_2 = CBF \times O_2 \text{ saturation} \times 1.31 \times \text{hemoglobin},$$

where O_2 saturation was obtained from a nomogram [9]. The constant 1.31 represents the O_2-carrying capacity of 1 g of hemoglobin [9]. Cerebral blood flow was corrected to a $PaCO_2$ of 40 mm Hg (CBF_{40}). The following formula was used:

$$CBF_{40} = CBF - k(PaCO_2 - 40),$$

where k is the measured CO_2 vasoreactivity (1.2 mL/100 g/min/mm Hg in this case).

Statistical Analysis

All values are given as the mean ± one standard deviation when not otherwise indicated. For comparison of values between groups, unpaired *t* tests were used according to RS/1 (Bolt, Beranek and Newman Inc, Cambridge, MA). One-way analysis of variance and paired *t* tests were used to evaluate the changes in CBF and $CDRo_2$ within the two groups. The significance level was a *p* value of less than 0.05. To compare CO_2 reactivity before, during, and after CPB, repeated-measures analysis of variance and covariance was done with BMDP (BMDP Statistical Software, Los Angeles, CA).

Results

One patient in the hypothermia group was found to have a right-sided hemiparesis 24 hours postoperatively. A computed tomographic scan revealed an infarction in the internal capsule area of the left hemisphere. All other patients had an excellent result as judged from clinical observations.

Basic Group Differences

Cerebral blood flow and related physiological variables under higher and lower $PaCO_2$ conditions determined before, during, and after CPB are shown in Table 2. There were no important intergroup differences except for a lower body temperature in the hypothermia group during CPB followed by a somewhat elevated temperature after CPB because of rewarming. In the normothermia group, body temperature was close to 34.5°C throughout the study.

In Table 3, the values corresponding to the various paired measurements are presented. The CBF and $CDRo_2$ (both corrected to a $PaCO_2$ of 40 mm Hg) are given.

Carbon Dioxide Reactivity

In both the normothermic and hypothermic patient groups, an increase in ventilation (before and after CPB)

Table 3. Cerebral Blood Flow, Cerebral Delivered Oxygen Rate, and Related Variables for the Two Patient Groups[a,b]

		Paired Measurements						
		Before CPB		During CPB		After CPB		
Variable	Group	1	2	3	4	5	6	p Value[c]
CBF_{40} (mL/100 g/min)	H	32 ± 10	30 ± 16	32 ± 11[d]	28 ± 7[d]	43 ± 10	33 ± 5	0.05
	N	25 ± 9	29 ± 9	42 ± 5	38 ± 5	40 ± 9	44 ± 15	0.001
External auditory temperature (°C)	H	34.9 ± 0.5	35 ± 0.6	28.8 ± 1.4[d]	28.8 ± 0.8[d]	36.8 ± 1.0[d]	35.6 ± 0.5[d]	0.05
	N	34.6 ± 0.5	34.5 ± 0.5	34.4 ± 0.7	34.7 ± 0.7	34.6 ± 0.5	34.5 ± 0.6	NS
Arterial blood pressure (mm Hg)	H	70 ± 22	68 ± 21	57 ± 22	70 ± 24	59 ± 19	67 ± 21	NS
	N	73 ± 16	69 ± 7	66 ± 15	76 ± 13	64 ± 11	67 ± 9	NS
Hemoglobin (g/L)	H	141 ± 14	144 ± 14	101 ± 13	106 ± 14	114 ± 17	115 ± 13	0.05
	N	137 ± 10	140 ± 12	98 ± 15	97 ± 10	101 ± 11	108 ± 8	0.05
PaO_2 (mm Hg)	H	123 ± 28	129 ± 30	160 ± 45[d]	126 ± 30	129 ± 75	138 ± 70	NS
	N	141 ± 28	135 ± 35	111 ± 36	109 ± 21	130 ± 40	126 ± 46	NS
$CDRo_2$ (mL/100 g/min)	H	5.6 ± 1.5	5.3 ± 2.4	4.1 ± 1.1[d]	3.8 ± 0.9[d]	6.2 ± 1.3	4.8 ± 0.7	0.008
	N	4.4 ± 1.5	5.2 ± 1.7	5.1 ± 0.4	4.7 ± 0.7	5.1 ± 0.8	6.0 ± 1.8	NS

[a] For all measurements, there were 9 patients in each group. Unlike Table 2, the data for measurement 1 represent results from the first measurement in all patients, and so on. See text for details. [b] Data are shown as the mean ± the standard deviation. [c] Values are from one-way analysis of variance. [d] Significance: $p < 0.05$ between the two groups.

CBF_{40} = cerebral blood flow at partial pressure of arterial carbon dioxide = 40 mm Hg; $CDRo_2$ = cerebral delivered oxygen rate at partial pressure of arterial carbon dioxide = 40 mm Hg; CPB = cardiopulmonary bypass; H = hypothermia; N = normothermia; NS = not significant; PaO_2 = partial pressure of arterial oxygen; arabic numerals 1 through 6 correspond to the individual measurements.

or a decrease in CO_2 administration (during CPB) caused a $PaCO_2$ decrease ($p < 0.05$) and a concomitant increase in pH. The resulting decrease in CBF was significant. Mean arterial blood pressure was not affected by hyperventilation. The cerebrovascular reactivity to $PaCO_2$, calculated as ΔCBF per $\Delta PaCO_2$, before, during, and after CPB is given in Table 4. The difference between the values obtained at normothermia or hypothermia was not significant, nor was the gradual increase in CO_2 vasoreaction from before to after CPB. The overall vasoreactivity, calculated as the mean of all measurements in both groups, was identical. In the normothermia group, ΔCBF per $\Delta PaCO_2$ was 1.2 ± 0.6 mL/100 g/min/mm Hg compared with 1.2 ± 0.8 mL/100 g/min/mm Hg in the hypothermia group.

Effect of Hemodilution

The effect of hemodilution on CBF (recalculated at 40 mm Hg) before and during CPB is illustrated in Figure 2A. Hemodilution in both groups was demonstrated by a decrease of about 40% in the hemoglobin concentration compared with the values seen before CPB (Fig 2B). In the normothermia group, there was a significant increase in CBF compared with the values measured before CPB ($p < 0.05$). The increase in CBF matched the decrease in hemoglobin concentration, so that $CDRo_2$ in the normothermic individuals remained unchanged (Fig 2D). In the hypothermia group (Fig 2C), hemodilution was not accompanied by an increase in CBF. As illustrated in Figure 3, a linear relationship between CBF (corrected for deviations from $PaCO_2$ of 40 mm Hg) and body temperature was found, thus explaining the absence of an increase in CBF during hypothermic CPB. The decrease in $CDRo_2$ (Fig 2D) of about 0.2 mL/100 g/min/°C was not significant.

Table 4. Carbon Dioxide Reactivity Before, During, and After Cardiopulmonary Bypass[a]

Time	Hypothermia Group	Normothermia Group	Both Groups
Before	0.8 ± 0.5	1.1 ± 0.6	0.9 ± 0.6
During	1.2 ± 0.8	1.2 ± 0.6	1.2 ± 0.7
After	1.5 ± 1.3	1.6 ± 1.2	1.6 ± 1.2

[a] Carbon dioxide reactivity is expressed as the cerebral blood flow change in milliliters per change in partial pressure of arterial carbon dioxide in millimeters of mercury.

Pattern of CBF and $CDRo_2$ Changes After CPB

Although the initial CBF values after CPB were almost identical for the two patient groups, a different flow pattern was observed for the second measurement after CPB (see Table 3). In the hypothermia group, CBF decreased by 23% from 43 to 33 mL/100 g/min, with a correlation to time after termination of CPB ($r = 0.5$, $p < 0.02$) (Fig 4). In the normothermia group, there was a minor increase in CBF.

In the hypothermia group, there was a transient increase in $CDRo_2$ immediately after CPB ($p < 0.05$), but the $CDRo_2$ returned to the level measured before CPB within 20 minutes. In contrast, $CDRo_2$ remained constant in the normothermia group throughout the observation period.

Comment

The cause and time of onset of the hemiparesis noted in 1 patient is unclear. We consider this complication as mac-

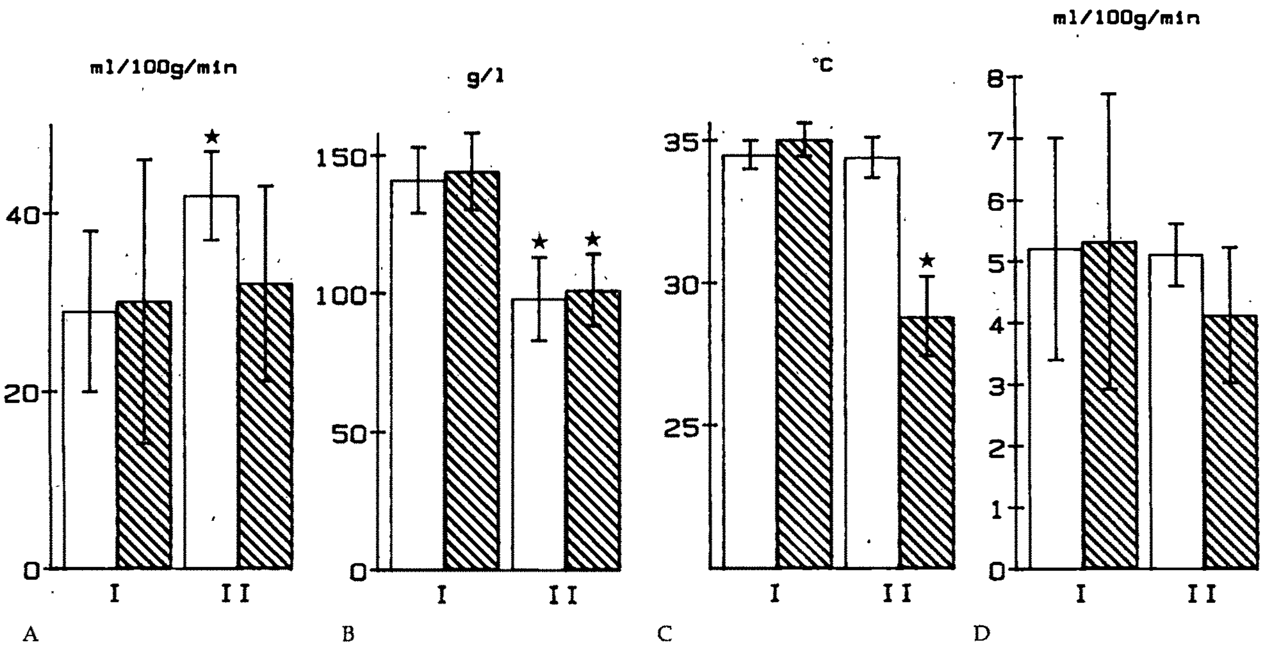

Fig 2. (A) Cerebral blood flow, (B) hemoglobin, (C) temperature, and (D) oxygen delivery rate to the brain for patients in the normothermia group (open bar) *and the hypothermia group* (hatched bar) *immediately before* (I) *and during* (II) *cardiopulmonary bypass.* (★ = $p < 0.05$.)

roembolic and possibly originating from the operative field. Because the onset seemed late, it is also possible that it occurred after CPB. The data for this patient do not imply a cerebral pathology during or immediately after CPB.

The results confirm previous reports of a maintained cerebral vasoreactivity to $PaCO_2$ changes during hypothermic nonpulsatile CPB with alpha-stat management. The reactivity, defined as ΔCBF per $\Delta PaCO_2$, was 1.2 mL/100 g/min/mm Hg during both hypothermic and normothermic CPB. Prough and colleagues [10] found a CO_2 reactivity during hypothermic CPB of 1.1 mL/100 g/min/mm Hg for the $PaCO_2$ range 36 to 42 mm Hg and 1.2 mL/100 g/min/mm Hg for the $PaCO_2$ range 47 to 53 mm Hg. Wollman and associates [11] had earlier arrived at a similar result by measuring the decrease in cerebral arteriovenous difference for O_2 when $PaCO_2$ was increased. Govier and co-workers [12] used a stepwise linear regression technique to show that CBF during hypothermic CPB was dependent mainly on temperature but also on $PaCO_2$.

In 1983, Henriksen and co-authors [13] presented results suggesting that cerebral vasoreactivity to CO_2 was decreased during hypothermic CPB with pH-stat management. However, this finding was reevaluated by Henriksen in 1986 [3]. He then found that $PaCO_2$ vasoreactivity during hypothermic CPB was maintained regardless of the existing $PaCO_2$ level. Lundar and colleagues [14] described preserved CO_2 reactivity in patients with pH-

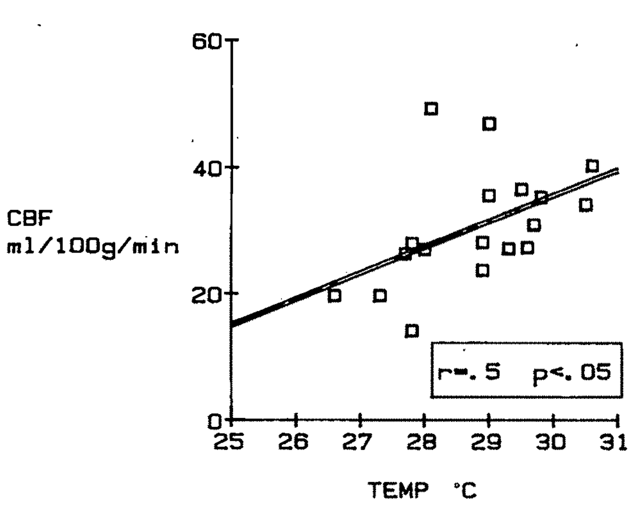

Fig 3. Relationship between temperature and cerebral blood flow (CBF) *during hypothermic cardiopulmonary bypass.*

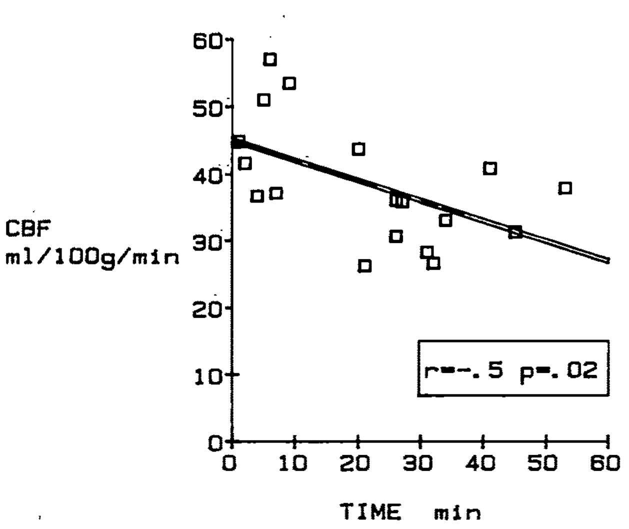

Fig 4. Relationship between cerebral blood flow (CBF) *and time passed after termination of hypothermic cardiopulmonary bypass.*

stat management using a transcranial Doppler method for assessment of CBF.

Thus, there is an increasing amount of evidence, including the present report, supporting the theory that CO_2 vasoreactivity is preserved during both normothermic and hypothermic CPB. The higher $PaCO_2$ levels during hypothermic CPB with the pH-stat perfusion method are widely used and are well tolerated by most patients, but apart from the evident risk of increased microembolism [3], the autoregulation [14, 15] and the coupling between flow and brain metabolism [16] are impaired with this method. With the alpha-stat regulation strategy, however, both autoregulation and flow–metabolism coupling are maintained, as was shown by Murkin and associates [16].

In our previous study [6], the CBF levels measured before and during hypothermic CPB were almost identical, presumably due to the fact that the CBF increase caused by hemodilution was counteracted by the hypothermia. These observations, which are supported by the present results, differ from the findings in other reports. Govier and associates [12] and Murkin and co-workers [16] found a decreased CBF during hypothermic CPB compared with the values seen before CPB. The lower CBF value during CPB correlated to the temperature decrease and the decrease in cerebral metabolic rate of oxygen ($CMRo_2$). The authors did not report on the effect of hemodilution on CBF.

In the present study, there was a significant ($p < 0.05$) correlation between CBF and body temperature during hypothermic CPB (see Fig 3). Our results also show that CBF was influenced by the decrease in O_2-carrying capacity caused by hemodilution, which is in agreement with the findings in other reports [17, 18].

To analyze the effect of hemodilution, CPB, and hypothermia on O_2 delivery to the brain, the $CDRo_2$ was calculated for a $PaCO_2$ of 40 mm Hg. The $CDRo_2$ ranged between 4 and 6 mL/100 g/min (see Table 3), which is about two times the $CMRo_2$ for normal awake subjects [19] and four times the $CMRo_2$ for patients at a comparable level of anesthesia [16]. In the hypothermia group, there was a decrease in $CDRo_2$ during hypothermia of about 0.2 mL/100 g/min/°C. Murkin and associates [16] found a $CMRo_2$ reduction during hypothermia of about 0.1 mL/100 g/min/°C. In the normothermia group, we found that the CBF increase during CPB responded to the decrease in O_2-carrying capacity as a result of hemodilution, so that the $CDRo_2$ remained unchanged.

Immediately after hypothermic CPB, there was a transient increase in CBF and $CDRo_2$, that was not entirely explained by the temperature difference and that normalized within 20 minutes. This transient CBF (and $CDRo_2$) increase after hypothermic CPB is similar to that found in our previous study [6] and may be due to washout of vasoactive substances accumulated during hypothermia. Govier and co-workers [12] also found a slightly, but significantly, increased CBF immediately after CPB.

The temporal behavior of CBF (and $CDRo_2$) after CPB in the normothermia group was significantly different from that in the hypothermia group, with a slight, nonsignificant increase with time. The present study does not provide sufficient information for a pathophysiological interpretation of this intergroup difference in CBF and $CDRo_2$. It cannot be explained by differences in $PaCO_2$, temperature, or degree of anesthesia. Perfusion time in the hypothermia group was longer, but the degree of CBF change was not correlated to perfusion time either in this study or the previous study [6].

A speculative interpretation is that the hypothermic patients had a 40% lower CBF than the normothermic patients during CPB, and thus a lower degree of exposure to the circulating microemboli that are known to occur during CPB [20]. In pigs, normothermic CPB resulted in an interruption of cerebral capillary blood flow in a study by Sörensen and associates [21], whereas hypothermic CPB did not lead to that result. According to that interpretation, the more heterogeneous and in some cases hyperemic blood flow levels in the normothermia group at the second measurement after CPB could be caused by reactive hyperemia after embolization.

An alternative interpretation of the present results is that the rather constant $CDRo_2$ level in the normothermia group throughout the operation represents the normal level at the given conditions, and that the decreasing CBF and $CDRo_2$ after CPB in the hypothermia group is a reaction to the hyperemia seen immediately after CPB. The hyperemia is caused either by an insufficient microcirculation and removal of cerebral waste products during the hypothermic phase with lower CBF and regionally collapsed capillaries [22] or by a washout of vasoactive substances from other parts of the rewarmed body.

In conclusion, this study shows that cerebral vasoreactivity to changes in $PaCO_2$ was maintained during normothermic and hypothermic CPB. In consequence of the preserved CO_2 vasoreactivity, the respiratory acidosis during hypothermic CPB with pH-stat management gives rise to a CO_2-mediated cerebral hyperemia that endangers autoregulation and uncouples flow and metabolism. The results corroborate those of previous reports indicating that alpha-stat management is the method of choice for hypothermic CPB. A comparison between hypothermic and normothermic CPB shows a significant difference in the CBF patterns after CPB between the two groups, but the pathophysiological interpretation of this finding remains unclear and needs further investigation.

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Use of Azygos Vein as Interposition Graft for Surgical Unifocalization of Pulmonary Blood Supply

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Hemodynamic repair in patients with pulmonary atresia, ventricular septal defect, hypoplastic pulmonary arteries with arborization defects, and major aortopulmonary collaterals necessitates prior unifocalization of pulmonary artery blood supply. When direct anastomosis between these collateral vessels and the central pulmonary arteries is not technically feasible, an interposition graft is required. Dacron, polytetrafluoroethylene, and pericardium have been used for this purpose. We describe our experience with the use of azygos vein as an interposition graft in 3 patients who underwent unifocalization of the pulmonary blood supply to the right lung. Our experience suggests that the azygos vein is a useful graft for use in this specific form of unifocalization procedure.

(Ann Thorac Surg 1989;48:776–8)

In patients with pulmonary atresia and ventricular septal defect complicated by arborization defects involving a substantial number of bronchopulmonary segments, complete hemodynamic repair is not feasible unless the pulmonary blood supply is "unifocalized" by surgical disconnection of these major aortopulmonary collateral arteries (MAPCAs) from the systemic circulation and anastomosis to the central pulmonary arteries. In most instances, adequate mobilization of the MAPCA facilitates direct anastomosis with the central pulmonary arteries. However, in situations in which insufficient length precludes direct anastomosis, an interposition graft is required. Dacron [1], polytetrafluoroethylene [2] and pericardial tubes [3] have been used for this purpose. We describe 3 patients in whom a segment of the azygos vein was used as a conduit to establish continuity between MAPCAs and the right pulmonary artery.

Case Reports

Patient 1

A 4-year-old girl was diagnosed at age 2 weeks as having pulmonary atresia, ventricular septal defect, hypoplastic pulmonary arteries with arborization defects, and MAPCAs and was admitted into a program for staged repair. In 1985, when the patient was aged 4 weeks, an end-to-side main pulmonary artery to ascending aorta shunt was performed. In 1987, through a left thoracotomy, a left pulmonary artery stenosis was repaired and two MAPCAs were ligated. In 1988, repeat angiography demonstrated two MAPCAs arising from a right descending thoracic aorta, supplying the right lung. Both were stenosed at the origin and did not communicate with the central pulmonary arteries. A right thoracotomy was performed, and the MAPCAs were identified as they entered the back of the hilum of the right lung. They were too short, however, for direct anastomosis to the pulmonary artery. The MAPCAs were disconnected from the descending thoracic aorta and the transected end of the superior MAPCA was anastomosed end-to-end with a 3-cm segment of azygos vein using a continuous 7-0 Prolene (Ethicon) suture. The other end of the vein segment was anastomosed end-to-side to the posterosuperior aspect of the right pulmonary artery. The second MAPCA was then anastomosed end-to-side to the inferior aspect of the first MAPCA. All anastomoses were made as long and as oblique as possible. Systemic saturation was 70% at the end of the procedure. An additional shunt procedure was contemplated at this stage but was deferred. The patient was transferred to the intensive care unit but returned to the operating room the next day as systemic saturations remained low. A right descending thoracic aorta to right pulmonary artery shunt was performed using a 5-mm Gore-Tex graft; the pulmonary anastomosis was performed proximal to the anastomosis with the azygos vein (Fig 1A). The patient has remained well since then and awaits recatheterization before definitive repair.

Patient 2

This patient was 18 months old at the time of MAPCA transplantation. He was diagnosed in infancy to have pulmonary atresia, a ventricular septal defect, hypoplastic pulmonary arteries, and MAPCAs. In addition, he had severe bronchial asthma. When he was aged 1 year, an end-to-side main pulmonary artery to ascending aorta shunt was performed using cardiopulmonary bypass. At recatheterization, a complex MAPCA arising from the right descending thoracic aorta and supplying both lungs was identified. Major aortopulmonary collateral artery transplantation was performed through a right thoracotomy. At operation, a double-barrelled takeoff of the

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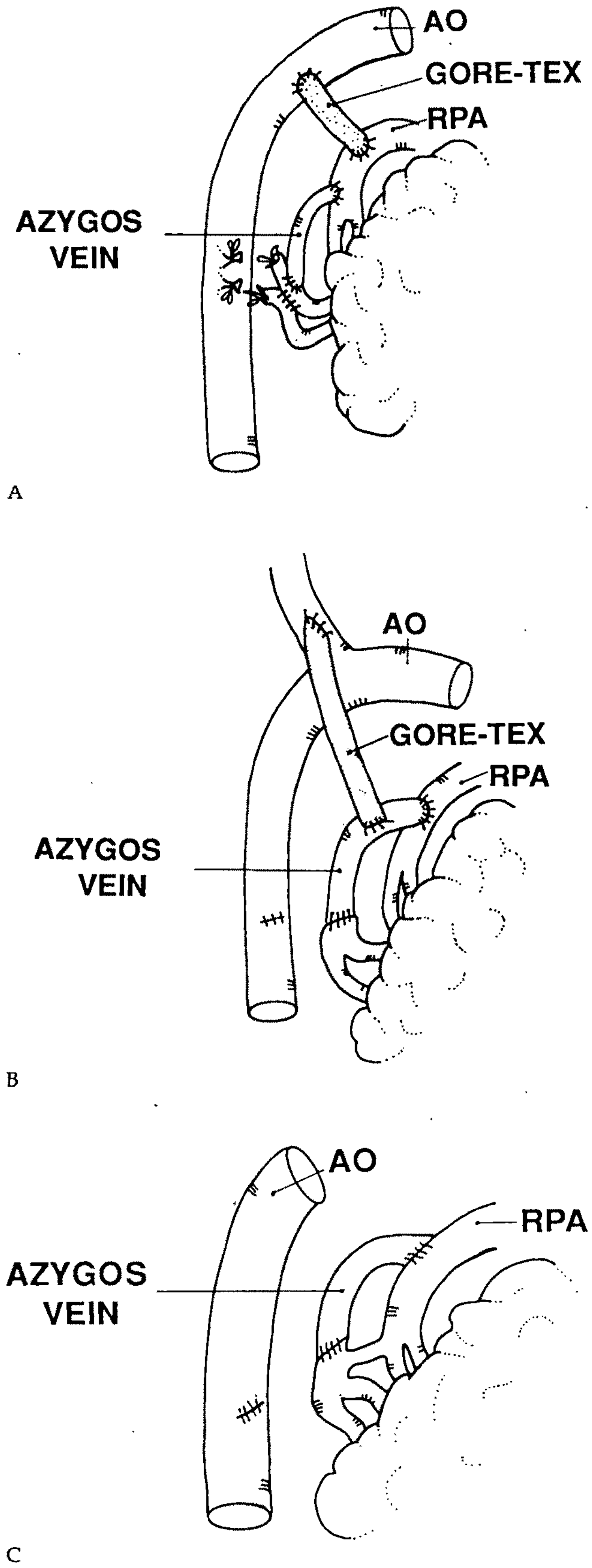

Fig 1. Azygos vein grafts were used in all 3 patients (A, patient 1; B, patient 2; C, patient 3). (AO = *aorta;* RPA = *right pulmonary artery.)*

complex MAPCA from the right descending thoracic aorta was identified with immediate branching to both lungs. When the right pulmonary artery was dissected out, no upper lobe branch could be found. The MAPCA was disconnected from the aorta and, as before, a segment of azygos vein was used to connect it to the right pulmonary artery. The patient had a stormy postoperative course with unstable hemodynamics and fluctuating systemic saturations. Two days later, a shunt was performed by interposing a 5-mm Gore-Tex graft between the right subclavian artery and the azygos vein graft (Fig 1B). The patient was then successfully weaned off the ventilator. He continues to suffer from severe asthma, but between episodes has good systemic saturation.

Patient 3

This patient was only 7 months old when he underwent the first-stage procedure for multistaged repair of pulmonary atresia, ventricular septal defect, and hypoplastic pulmonary arteries. Through a left thoracotomy, an end-to-side main pulmonary artery to ascending aorta shunt was performed and one minor aortopulmonary collateral artery was ligated. He was readmitted 3 weeks later with congestive cardiac failure, and a decision was made to proceed with the second stage of repair. Through a right thoracotomy, a right descending thoracic aorta was identified, as were two large MAPCAs arising close together immediately behind the hilum of the right lung. The MAPCAs were clearly too short for direct anastomosis to the right pulmonary artery and instead the azygos vein had to be used as an interposition graft (Fig 1C). The patient remained in cardiac failure postoperatively, and repeat cardiac catheterization showed that the left pulmonary artery had grown from 3 to 8 mm and the right pulmonary artery had grown from 2 to 5 mm in just 4 weeks. The mean pulmonary artery pressure measured 25 mm Hg with a pulmonary to systemic blood flow ratio greater than 2:1. Definitive repair was therefore immediately performed, and the right pulmonary artery was extensively reconstructed with autologous pericardium, the VSD was closed, and right ventricle to main pulmonary artery continuity was established with a 14-mm Hancock conduit. The anastomosis of the azygos vein to the right pulmonary artery was inspected from within and was widely patent. The patient made an uneventful recovery from operation and underwent repeat cardiac catheterization and angiography 1 month postoperatively. The systolic pressure ratio of the right ventricle to the left ventricle was 0.5, and the azygos vein conduit filled nicely, with no evidence of stenosis (Fig 2).

Comment

The combination of pulmonary atresia, ventricular septal defect, hypoplastic pulmonary arteries with arborization defects, and MAPCAs comprises a subgroup of congenital heart disease that is difficult to repair surgically. In our experience, the best results have been obtained with staged repair, which involves performing a central shunt, unifocalizing the pulmonary blood supply, ligating minor aortopulmonary collateral arteries, and finally performing

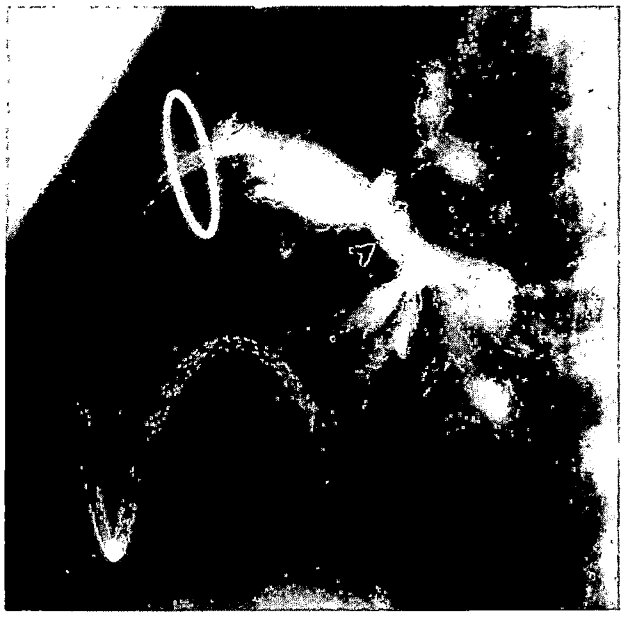

Fig 2. Postoperative right pulmonary artery angiogram in right lateral projection showing patent azygos vein graft (arrowhead) *in patient 3.*

hemodynamic repair after satisfactory growth of the pulmonary arteries has been achieved. Unifocalization is an important step in this staged procedure because it achieves incorporation of the maximum number of bronchopulmonary segments into the central pulmonary artery supply. The resultant increase in flow through the central pulmonary artery promotes accelerated growth. In addition, the MAPCAs may be anastomosed across areas of narrowing in the central pulmonary artery, thereby widening those areas.

Although a direct anastomosis between the MAPCAs and the central pulmonary arteries is ideal, it is not always feasible. Prosthetic conduits have the inherent disadvantage of low long-term patency rates [2], especially in the smaller sizes and in low-pressure conditions, as may be expected in a branch pulmonary artery. The azygos vein is an appealing alternative in that it is autologous and therefore presumably less thrombogenic and generally conforms to the caliber of the MAPCAs requiring transplantation. In addition, the azygos vein graft could grow concomitantly with the rest of the pulmonary vascular bed. However, the azygos vein can be used only in the right side of the chest. That all 3 patients we describe had a right descending aorta with short-stemmed MAPCAs arising directly behind the right hilum underscores the recurrent patterns seen with MAPCAs.

Early patency has been angiographically documented in at least 1 of our patients. The other 2 are awaiting definitive repair; however, their clinical condition suggests that their grafts are still patent. Estimates of long-term patency are conjectural in the absence of long-term follow-up; nevertheless, the azygos vein appears to be an appropriate graft for use in this rather specific form of unifocalization procedure.

Addendum

Subsequent to the submission of the manuscript, 1 more patient with a right descending thoracic aorta and similar collateral anatomy underwent MAPCA transplantation using azygos vein as an interposition graft.

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Surgical Treatment of Pulmonary Aspergilloma and *Aspergillus* Empyema

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Twenty-four patients, 16 male and 8 female, underwent a total of 35 operations for pulmonary aspergillosis. Intrapulmonary aspergilloma was detected in 19, and *Aspergillus* empyema was present in 5. The major operative procedures performed were pneumonectomy in 2 patients, lobectomy in 8, segmentectomy in 1, cavernostomy in 4, and open-window thoracostomy in 5. The surgical results in 5 patients 70 years old or older were excellent. Empyema developed postoperatively in 2 patients who had undergone wedge resection of the lung or segmentectomy. Although resection involving the minimum extent possible is desirable in the treatment of intrapulmonary aspergilloma so as not to decrease lung function, it is dangerous to perform a limited procedure in the case of aspergilloma with an invasive character. In patients in poor general condition, cavernostomy followed by muscle flap plombage is recommended. For *Aspergillus* empyema, the primary procedure was open-window thoracostomy followed by plombage using chest wall muscle or omentum. We consider omental flap plombage to be superior to thoracoplasty in some respects for mycotic empyema, especially because it is a less extensive surgical procedure.

(Ann Thorac Surg 1989;48:779–82)

Among mycotic diseases of the lung, pulmonary aspergillosis is the one encountered most frequently in Japan. Human pulmonary aspergillosis is clinically divided into primary and secondary forms. Secondary aspergillosis is usually found in damaged lungs or in the lungs of a host with impaired defense mechanisms. In 1970, Zimmermen and Miller [1] classified pulmonary aspergillosis as follows: (1) secondary noninvasive (mycetoma); (2) secondary invasive; (3) primary invasive; and (4) primary allergic. It had been thought that *Aspergillus* mycetoma usually grew in preexisting cavities in the lungs, such as old tuberculous or bronchiectatic cavities. However, in 1981 Gefter and colleagues [2] proposed another type of aspergillosis, a "semi-invasive" type, after observations suggesting that mycetoma might be produced by *Aspergillus* in the absence of previous cavitary lung disease [3]. Daly and associates [4] classified pulmonary aspergillosis roentgenographically as simple type and complex type, where simple type means noninvasive or saprophytic and complex type means invasive.

The most frequent and important symptom of aspergilloma, whether noninvasive, semi-invasive, or invasive, is hemoptysis [3–6]. For this reason, surgical treatment has been adopted because bleeding from such lesions can be fatal [7]. The operative procedures differ for intrapulmonary aspergilloma and *Aspergillus* empyema. This report reviews our recent experience with the surgical treatment of these two conditions.

Material and Methods

Patient Population

Between 1979 and 1987, 24 patients underwent 35 operative procedures for pulmonary aspergillosis. There were 16 men and 8 women whose age ranged from 22 to 76 years; almost half of them were in their sixth or seventh decade (Fig 1). The mean age was 58 years, and 5 patients were 70 years old or older. The diagnostic methods included routine chest roentgenograms, chest tomograms, computed tomography, and bacteriological examination of either sputum or aspirated material. In those patients with the characteristic roentgenographic appearance of mycetoma, the identification of a so-called fungus ball was considered the most important factor in making the diagnosis.

In 18 (75%) of the 24 patients, fungus balls were identified on the chest roentgenograms. Before operation, the diagnosis of aspergillosis was confirmed in 7 patients from the culture of specimens obtained by percutaneous aspiration or bronchofiberscopic lavage (sputum culture, 2 patients or 8.3%; transbronchial lavage or biopsy, 5 patients or 20.8%). The *Aspergillus* skin test was not routinely performed. The complement-fixation test for serum antibodies against *Aspergillus fumigatus* was positive in 6 patients (25%). Other methods were used in 3 patients (12.5%).

Hemoptysis was present in 14 patients (58%), and 5 patients complained of purulent sputum. Three patients were asymptomatic (Table 1). The underlying pulmonary diseases included tuberculosis in 13 patients and cystic lung lesions in 2 patients. Three of the patients had undergone previous operation for pulmonary tuberculosis, and aspergillosis had developed in the residual pleu-

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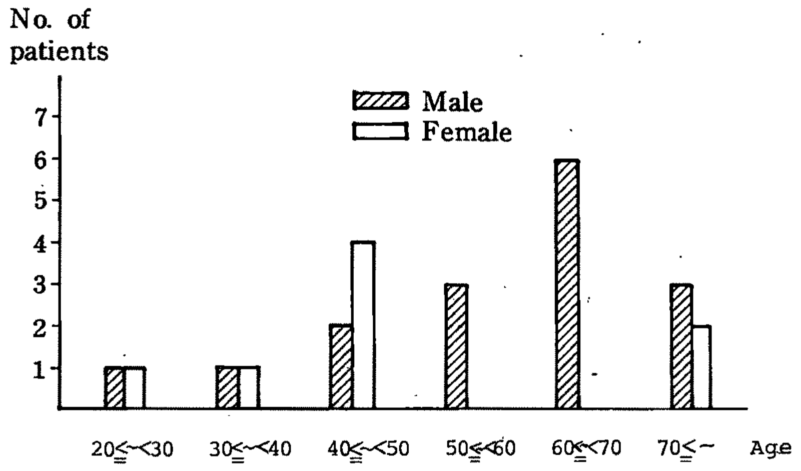

Fig 1. Age distribution of the patients with pulmonary aspergillosis.

ral space. The antifungus drug, 5-FC, was administered preoperatively to 4 patients.

Operative Procedures

The operative procedures are shown in Table 2. Lobectomy was the most frequent initial procedure for intrapulmonary aspergilloma. Wedge resection of the lung was performed in patients with either small peripheral lesions or poor respiratory functional reserve. Cavernostomy also was selected when the patient was in poor general condition. Open-window thoracostomy was carried out in 5 patients with intrapleural aspergilloma that had caused a thoracic empyema to develop. One patient underwent an exploratory thoracotomy because he was known to have a carcinomatous lesion, which was inoperable, in another lobe. All patients who underwent open-window thoracostomy or cavernostomy received daily changing of gauze and local instillation of amphotericin B. This was continued for 3 to 6 months, followed by plombage with an omental flap in 1 patient and muscle flap plombage in 5 to close the defects (see Table 2).

Table 1. Major Symptoms and Underlying Diseases in 24 Patients With Pulmonary Aspergillosis[a]

Variable	No. of Patients
Symptom	
Hemoptysis	14 (58.3)
Purulent sputum	5 (20.8)
Fever	2 (8.3)
Weight loss	2 (8.3)
None	4 (16.7)
Underlying disease	
Pulmonary tuberculosis	17 (58.3)[b]
Lung cyst	2 (8.3)
Previous lung carcinoma	2 (8.3)
Bronchiectasis	1 (4.2)
Unknown	1 (4.2)

[a] Numbers in parentheses are percentages. [b] Three of these patients had undergone surgical intervention previously for pulmonary tuberculosis.

Postoperatively, cultures of the intracavitary material from 18 patients grew *A fumigatus*. Histologically, *Aspergillus* was discovered in all resected specimens, which were stained with periodic acid–Schiff.

Results

There were no operative deaths. Twenty-two of the 24 patients had an uneventful recovery; complications developed in only 2 patients. A 32-year-old woman with aspergilloma in the right apex underwent wedge resection of the lung. Postoperatively, mycotic empyema with multiple fistulas developed in the residual space. Open-window thoracostomy was performed, and instillation of amphotericin B was undertaken daily for 3 months. Thereafter, the omentum was passed through the diaphragm and wrapped over the surface of the exposed lung, successfully closing the fistulas. Muscle flap plombage was also performed. Consequently the dead space was decreased, and the symptoms resolved completely.

The other patient with complications postoperatively underwent segmentectomy. An empyema developed, and open-window thoracostomy was performed. The patient subsequently died.

There were 3 late deaths. One patient died of a myocardial infarction, and 2 died of lung carcinoma. None died of aspergillosis (Table 3).

Comment

Pulmonary aspergillosis, particularly the intracavitary type, is a potentially life-threatening disease because it can cause sudden and massive hemoptysis [6–9]. When considering the mortality due to hemoptysis, a wide variation is seen [10]. Among patients whose underlying disease is a nontuberculous chronic lung disease, the mortality rate is low, but fatal hemorrhage is common when the underlying disease is pulmonary tuberculosis [7].

A definite diagnosis of aspergilloma is usually established by the characteristic appearance of a fungus ball on

Table 2. Operative Procedures in 24 Patients With Pulmonary Aspergillosis

Initial Operation	No. of Patients	Postop Empyema	Second Operation	Third Operation
Lobectomy	8	0	. . .	. . .
Segmentectomy	1	1	Open-window thoracostomy[a]	. . .
Wedge resection	5	1	Open-window thoracostomy	Omental flap plombage
Cavernostomy	4	0	Pneumonectomy (1); muscle flap plombage (2); omental flap plombage (1)	. . .
Open-window thoracostomy	5	0	Pneumonectomy (1); muscle flap plombage (3); other (1)[a]	. . .
Exploratory thoracotomy	1[a]	0	. . .	. . .

[a] This patient died late of other disease.

the chest roentgenogram, tomogram, or computed tomogram [11, 12]. Preoperative cultures of sputum are not always positive, though occasionally, cultures of either bronchofiberscopic lavage fluid or percutaneously aspirated material grow the organism.

The mortality rate in the early postoperative period was reported to be roughly 9% by Garvey and co-workers [13], 7% by Stamatis and Greschuchna [14], and 5% in 21 patients with simple aspergilloma and 34% in 32 patients with complex aspergilloma by Daly and colleagues [4]. Stamatis and Greschuchna [14] insisted that all good-risk patients, even if asymptomatic, should undergo lung resection because of the possibility of exsanguinating hemorrhage. However, pulmonary aspergilloma is essentially a benign disease, so the risk of early postoperative death should be completely avoidable.

Some authors [13, 14] believe that systemic medical therapy is largely unsuccessful, but in a series of 31 patients with aspergilloma and advanced lung disease, Faulkner and associates [6] were able to control hemorrhage without surgical intervention. Also, Hargis and coauthors [15] reported success with intracavitary instillation of amphotericin B in 6 patients with symptomatic pulmonary aspergillosis. In these patients, surgical resection was not feasible because of their past general condition. We [16] believe that in patients with poor pulmonary function or severe chronic obstructive lung disease, local instillation of an antimycotic agent is the treatment of choice [17].

Table 3. Operative Outcome for 24 Patients With Pulmonary Aspergillosis

Postop Period (yr)	No. of Patients	Outcome
0–2	7	2 died of other disease; 5 alive without recurrence
2–4	9	2 died of other disease; 7 alive without recurrence
4–6	3	3 alive without recurrence
≥6	5	5 alive without recurrence

Generally, when the following criteria are satisfied, surgical removal is recommended:

1. The aspergilloma and underlying lung disease are not widespread.
2. The patient has episodes of recurrent hemoptysis or chronic production of purulent sputum.
3. The patient is in relatively good condition and is able to tolerate a surgical procedure.

In elderly patients, removal of aspergilloma is probably indicated. Recently in Japan, the average life expectancy of the older generation has greatly increased, and the frequency of surgical treatment of various diseases in older patients is therefore increasing. In our series, we performed operations in 4 patients 70 years old or older and obtained good results. Compared with the age distribution in reports on aspergilloma from 10 to 20 years ago [5, 13, 18], an extension to the older generation is characteristic.

Garvey and associates [13] wrote that the extent of resection of intrapulmonary aspergilloma should depend on the severity of the underlying lung pathology as well as the aspergilloma itself. We believe that because of the saprophytic character of the organism, it is desirable to limit the resection as much as possible so as not to decrease lung function. Also, because the organism can invade the surrounding lung parenchyma, a segmental or wedge resection can sometimes be dangerous. In patients who are in poor general condition, cavernostomy followed by plombage using a muscle or omental flap is recommended because it has less effect on lung function and thus is not as hard for the patient to tolerate. We performed cavernostomy in 3 elderly patients with good results.

In 5 patients with *Aspergillus* empyema, we performed open-window thoracostomy followed by daily insertion of gauze impregnated with amphotericin B. In 3 of them, this was followed by plombage using a muscle flap, and the outcome was good in each patient. Stamatis and Greschuchna [14] have stated that for patients with pleural aspergillosis or mycotic empyema, only aggressive resection, that is, pneumonectomy, can provide effective long-term palliation. Battaglini and co-workers [19] also were of that opinion. However, we think that pneumonectomy should be performed only when there is substantial diffuse destruction of the lung, and when the extent of the aspergilloma and the underlying pulmonary disease have been considered.

In patients with an invasive or pyogenic component, there exists the possibility of postoperative bronchopleural fistula and empyema. In a series of 22 patients having resection reported by Aslam and co-workers [18], this complication occurred in 5 patients (23%). Saab and Almond [5] described complications in 11 (52%) of 21 patients. In the report by Daly and colleagues [4], postoperative complications were seen in 78% of patients with complex aspergilloma and 33% of patients with simple aspergilloma. We had two instances of postoperative local empyema, and both were caused by the use of a limited procedure (wedge resection or segmentectomy). It is important to carry out a sufficiently wide resection (lobectomy) in patients whose disease has invaded the surrounding lung parenchyma. In most patients, the postoperative complications, which include space problems or empyema [13], can be managed successfully in a standard fashion, such as prolonged chest tube drainage or thoracoplasty. In 1 of the 2 patients with postoperative empyema in this series, plombage using an omental flap was performed successfully. The omentum has a sufficient blood supply to occlude the bronchopleural fistulas produced by the invasion of *Aspergillus* organisms. We believe that the omental flap is useful in the treatment of mycotic empyema, especially when it occurs postoperatively.

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Effect of Transannular Patching on Outcome After Repair of Tetralogy of Fallot

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Among 814 patients undergoing repair of tetralogy of Fallot with pulmonary stenosis between 1967 and May 1986, transannular patching in the current era was a weak risk factor for death early postoperatively (predicted 30-day mortality, 4% with a transannular patch and 1.4% without) and is not a risk factor for instantaneous risk of death late postoperatively (predicted 20-year survival including early death, 94% with a transannular patch and 96.5% without). Ninety-six percent of surviving patients were in New York Heart Association functional class I at last follow-up, and the slight decline in this percentage as the interval between operation and last follow-up lengthened could have been due to chance alone (p = 0.24) and was no different in patients with a transannular patch. Transannular patching was a risk factor for reoperation for pulmonary regurgitation late postoperatively, but only a 7% incidence within 20 years is predicted when mild residual stenoses are beyond the patch: the incidence rises to about 20% with important distal stenoses. Inferences from the study are relevant to the indications for transannular patching and insertion of allograft semilunar valves at the time of repair.

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Controversy has continued over the indications for transannular patching as a part of the repair of tetralogy of Fallot with pulmonary stenosis, in large part because of insufficient information on the potentially detrimental effect on outcome of the resulting pulmonary regurgitation. Therefore a study has been made of the effect of transannular patching in the repair of tetralogy of Fallot on early and intermediate-term survival, functional status, and freedom from reoperation. The data are also relevant to the recent upsurge of interest in using semilunar valve allografts rather than transannular patches when the pulmonary annulus is hypoplastic.

Material and Methods

Patient Population

Between January 1, 1967, and May 1, 1986, 814 patients underwent repair of tetralogy of Fallot with pulmonary stenosis. Age at operation ranged between 2 months and 57 years. Ten percent of the patients were 1.0 year of age or less at operation, 25% were 2.3 years of age or less, 50% were 5.3 years of age (median age) or less, and 75% were 11 years of age or less.

The event "reoperation" included all reoperations early and late after the repair and excluded only reoperations for bleeding in the perioperative period. Patients who underwent percutaneous balloon valvoplasty of the pulmonary valve or angioplasty of the pulmonary trunk or right or left pulmonary artery stenoses were included among those considered to have undergone reoperation. The event was tabulated whether the reoperation was performed in this institution (28 reoperations, 1 hospital death) or elsewhere (4 reoperations, 1 hospital death). During the same period, 34 additional reoperations were performed in this institution (1 hospital death) in patients whose initial repair had been done elsewhere.

Intraoperative Methods

The surgical methods used [1] and the indications for placing a transannular patch [2] have been described previously. The repair was performed from the right atrium or by a combined right atrial and pulmonary arterial approach in 107 patients [3–5] and through a right ventriculotomy in 707. A simple repair (infundibular dissection, pulmonary valvotomy, or both, with or without an enlarging patch limited to the right ventricle, the pulmonary trunk, or both) was used in 538 patients, a transannular patch (or a nonvalved conduit, 2 patients) in 255 (31% of 814 patients), and a valved conduit in 21. Throughout the experience, the transannular patch was tailored to be of adequate width but not billowing. Most of the patches were made of Dacron, but pericardial patches were used early in the experience in some patients and throughout the experience in small patients.

The postrepair ratio between the peak pressure in the right ventricle and that in the left ventricle (postrepair $P_{RV/LV}$) was measured in the operating room (postrepair (OR) $P_{RV/LV}$) about 20 minutes after the discontinuance of cardiopulmonary bypass. A thin-walled needle was passed across the free wall and into the right ventricle; after the right ventricular peak pressure was obtained, the needle was advanced across the ventricular septum and into the left ventricular cavity. In general, a simple repair

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was used when the predicted postrepair (OR) $P_{RV/LV}$ was 0.85 or less. A transannular patch usually was placed if the predicted or measured ratio without a patch was greater than this [6].

Study Methods

All documents pertaining to the original operation and the postoperative period, all documents concerned with interval events including reoperations performed at this institution or elsewhere, and all information obtained directly from patients and their physicians were studied in detail. The pertinent information was extracted and placed in computer storage.

Follow-up

A formal follow-up of all patients not known to be dead was made by letter and by telephone during July, August, and September 1986. All but 68 patients were traced during follow-up or were previously known to be dead. Of the 68, 10 had 10 to 16 years of complete follow-up as a result of previous inquiry, return visits, correspondence, or a combination of these, 17 had 5 to 10 years, 18 had 2 to 5 years, 4 had 1 year to 2 years, 8 had 30 days to 1 year, and 11 had less than 30 days. The mean follow-up after initial repair was 9 ± 5.4 years. Ten percent of the patients were followed 16 or more years; 25%, 14 or more years; 50%, 10 (median follow-up) or more years; 75%, 4.4 or more years; and 95%, 1 year or more.

Analytical Methods

The time-related freedom from death was estimated actuarially and by parametric methods [7, 8]. The hazard function was derived using a method previously described [8]. Multivariate analyses were performed in the hazard function domain to search for incremental risk factors [8]. A variable was retained as a risk factor if its p value was less than 0.1. The time-related freedom from reoperation of any kind and the time-related freedom from reoperation for pulmonary regurgitation were similarly analyzed, but patients receiving valved conduits (mostly xenograft valved) were excluded. Ordinal logistic regression analysis was used to estimate parametrically the change in New York Heart Association functional class across time [9].

All differences were examined by appropriate methods for the likelihood that they were due to something other than chance alone.

Results

Survival

Transannular patching was a risk factor for death early after repair of tetralogy of Fallot but not thereafter (Table 1). The effect is much weaker in operations performed in recent years, and the trends suggest that currently any differences could be due to chance alone (Fig 1).

Functional Class Late Postoperatively

Ninety-six percent of surviving patients were in New York Heart Association functional class I at the time of the last follow-up. A slightly reduced prevalence of patients in class I when there was a longer interval between operation and last follow-up could have been due to chance alone (Fig 2). Thus, there are no risk factors (including transannular patching) for declining functional status in surviving patients (Appendix Table B-1).

Table 1. Incremental Risk Factors for Death After Repair of Tetralogy of Fallot With Pulmonary Stenosis[a,b]

Incremental Risk Factor for Death (All Deaths = 81)	Hazard Phase	
	Early	Constant
Demographic		
Body surface area (m^2) (smaller)	Yes	No
Age (yr) (older)	Yes	Yes
Clinical		
Hematocrit (%) (higher)	Yes	No
Previous Potts' anastomosis	No	Yes
More than one previous palliative operation	Yes	No
Previous palliative direct relief of RVOTO	No	Yes
Morphological		
Absence of unbranched hilar portion of one pulmonary artery	Yes	No
Multiple ventricular septal defects	Yes	No
Dextrocardia	Yes	No
Surgical		
Date of operation (years since 1/1/67) (earlier)	Yes	No
Transannular patch	Yes	No
Postrepair (OR) $P_{RV/LV}$ (higher)	Yes	Yes

[a] Variables with no notation in parentheses as to the direction or units of the risk factor are simply yes/no variables. [b] See Appendix A for the variables entered into the analysis and the parameter estimates, coefficients, and p values.

OR = operating room; $P_{RV/LV}$ = ratio between peak pressure in right ventricle and that in left ventricle; RVOTO = right ventricular outflow tract obstruction.

Reoperations

Transannular patching was not a risk factor for reoperation in general after repair of tetralogy of Fallot (Table 2). Transannular patching clearly was a risk factor for reoperation for pulmonary regurgitation (Fig 3). However, the strength of its effect was dependent on the postrepair (OR) $P_{RV/LV}$ (Table 3), and the probability of reoperation within 20 years was very low when residual stenosis was mild (Fig 4). Thus, transannular patching rarely resulted in reoperation for pulmonary regurgitation within 20 years when an obstructing lesion beyond the transannular patch was not present (Table 4).

Comment

Critique of Study

A strength of the study is that two of the outcome events, death and reoperation, are specific and are objectively

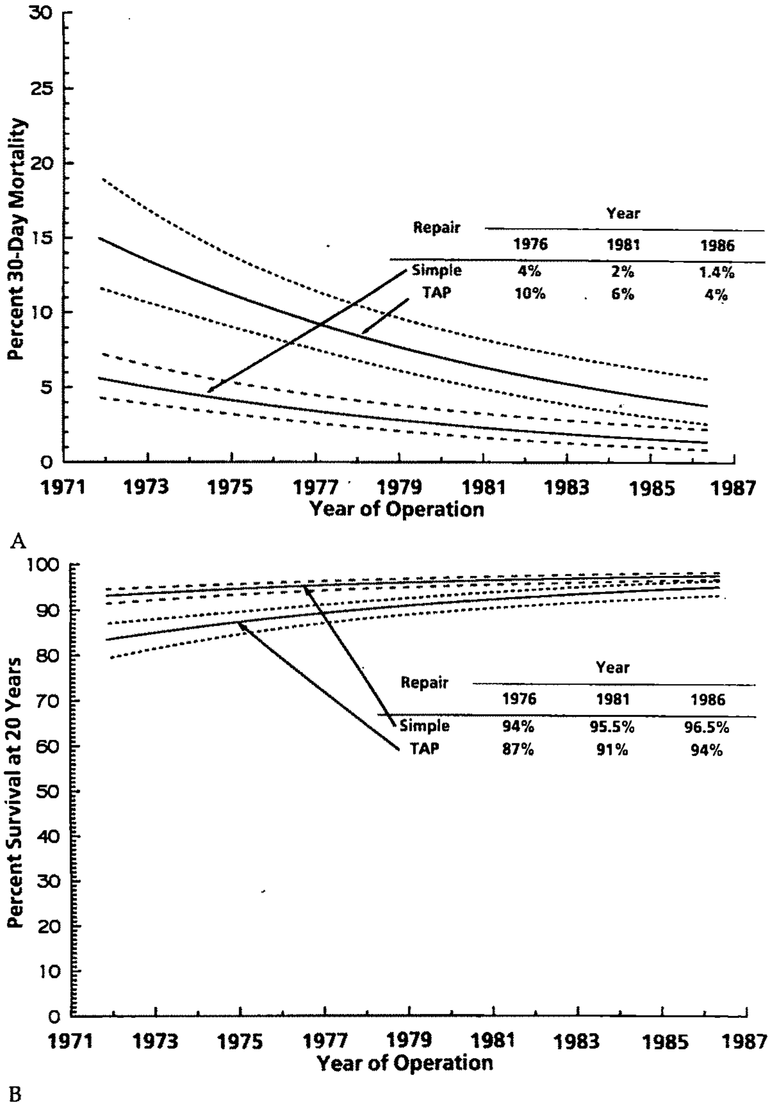


Fig 1. The effect on (A) 30-day mortality and (B) 20-year survival of use of a transannular patch (TAP) *rather than a simple procedure (dissection or valvotomy, with or without infundibular or pulmonary artery patching). Time zero is the time of operation, and thus hospital deaths are included. Patients 2 years of age at repair are represented. The* solid lines *are the continuous point estimates, and the* broken lines *enclose the 70% confidence intervals. Notice that the 70% confidence intervals of (A) death and (B) survival are nearly touching when the repair, whether simple or with a patch, was done in 1986, thus indicating that the difference by that date may be due to chance alone. The depictions are specific solutions of the multivariate risk-factor equation shown in Table 1 (see Appendix A for details).*

determined. The third, New York Heart Association functional class, is specific but is subjectively determined, and on occasion, patients who believe themselves to have no limitation on their activities are found to be subnormal by objective exercise testing. However, in the setting of the current study, the data concerning the functional class over a long follow-up would seem to be useful, particularly as there is no evidence out to 20 years of deteriora-

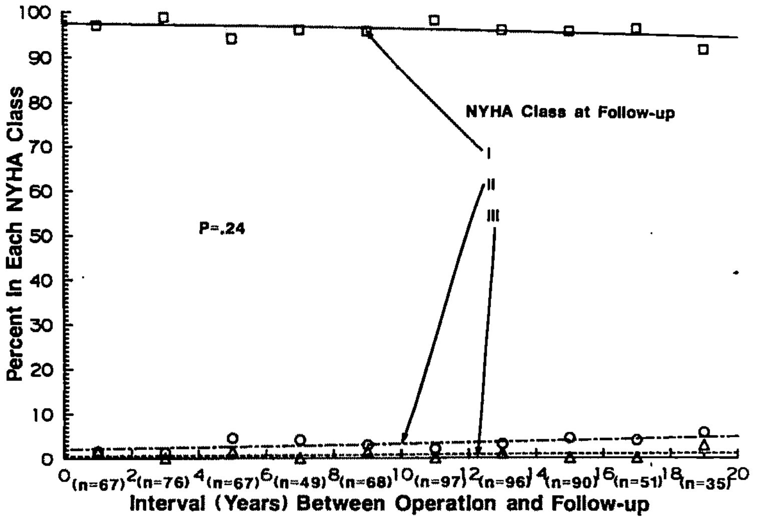


Fig 2. New York Heart Association (NYHA) *functional class after repair of tetralogy of Fallot according to the interval between operation and last follow-up. The numbers in parentheses along the horizontal axis are the number of patients available for functional class categorization at the odd-numbered interval below which the number is positioned. The* squares *represent the actual proportion of patients in functional class I at each interval; the* circles, *those in class II; and the* triangles, *those in class III. The* solid line *represents the actual slope of the solution of the ordinal logistic regression analysis, although the p value of 0.24 for the difference from zero slope indicates that change across time in the distribution of patients according to their NYHA functional class could be due to chance alone (see Appendix B for details).*

Table 2. Incremental Risk Factors for Reoperation of Any Type After Repair of Tetralogy of Fallot[a,b]

	Hazard Phase	
Incremental Risk Factor for Reoperation of Any Type (n = 32)	Early p value	Constant p value
Demographic		
Body surface area (m^2) (smaller)	No	Yes
Previous operation		
Potts' anastomosis	No	Yes
Palliative direct relief of RVOTO	No	Yes
Surgical		
Valved extracardiac conduit	No	Yes
Postrepair (OR) $P_{RV/LV}$ (higher)	Yes	Yes

[a] The depiction is as in Table 1. [b] See Appendix C for details.

OR = operating room; $P_{RV/LV}$ = ratio between peak pressure in right ventricle and that in left ventricle; RVOTO = right ventricular outflow tract obstruction.

Table 3. Incremental Risk Factor for Reoperation for Important Pulmonary Regurgitation After Repair of Tetralogy of Fallot[a]

Incremental Risk Factor for Reoperation for Important Pulmonary Regurgitation (n = 5)	Single Hazard Phase p value
Postrepair (OR) $P_{RV/LV}$ (higher)	0.1

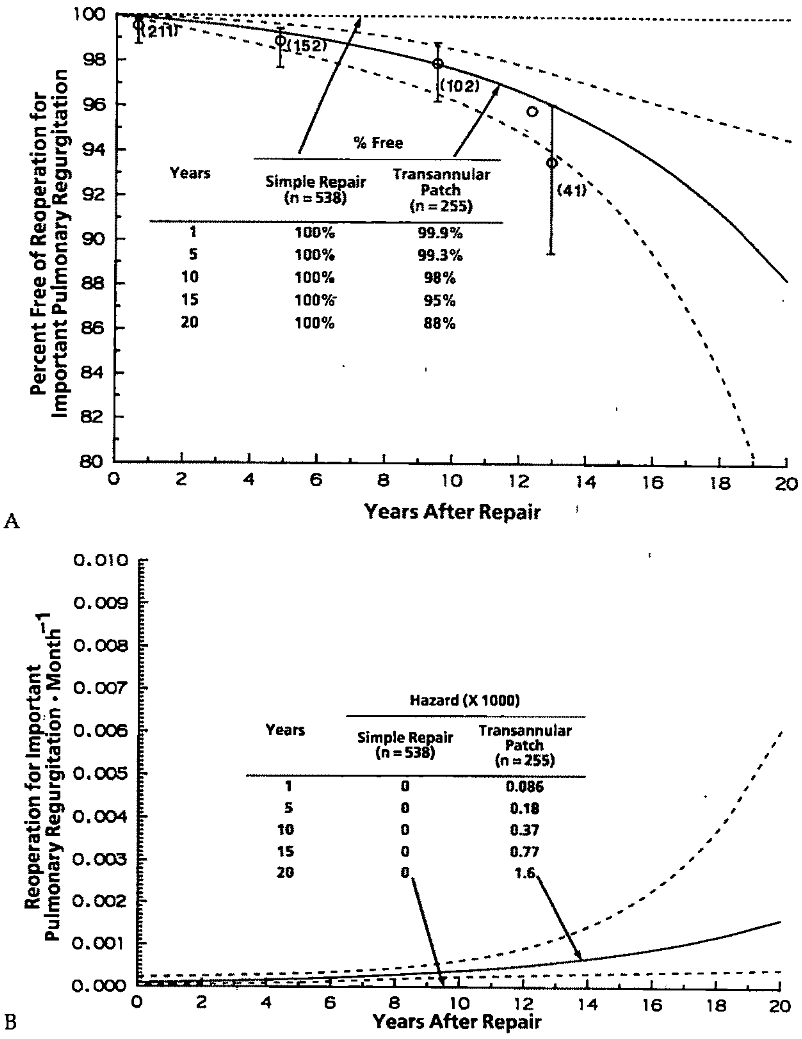
[a] See Appendix D for details.

OR = operating room; $P_{RV/LV}$ = ratio between peak pressure in right ventricle and that in left ventricle.

tion in the functional capabilities of the patients. The reliability of the outcome data is enhanced by the completeness and length of the follow-up.

Ideally, late postoperative hemodynamic and objective exercise testing should be included in the outcome events studied for the determination of a possible unfavorable effect of transannular patching. These data are, however, almost impossible to obtain on anything more than a small fraction of the patient population, and this severely

Fig 3. *(A) Actuarial and parametric depiction of percent freedom from reoperation for important pulmonary regurgitation according to whether the repair was simple or was performed using a transannular patch. Among the patients who underwent simple repair, no reoperations were performed for important pulmonary regurgitation, and this is shown by the* straight broken line. *Note the markedly expanded vertical axis. (B) Hazard function (see Appendix C for details).*



Years	% Free Simple Repair (n = 538)	% Free Transannular Patch (n = 255)
1	100%	99.9%
5	100%	99.3%
10	100%	98%
15	100%	95%
20	100%	88%

Years	Hazard (X 1000) Simple Repair (n = 538)	Hazard (X 1000) Transannular Patch (n = 255)
1	0	0.086
5	0	0.18
10	0	0.37
15	0	0.77
20	0	1.6

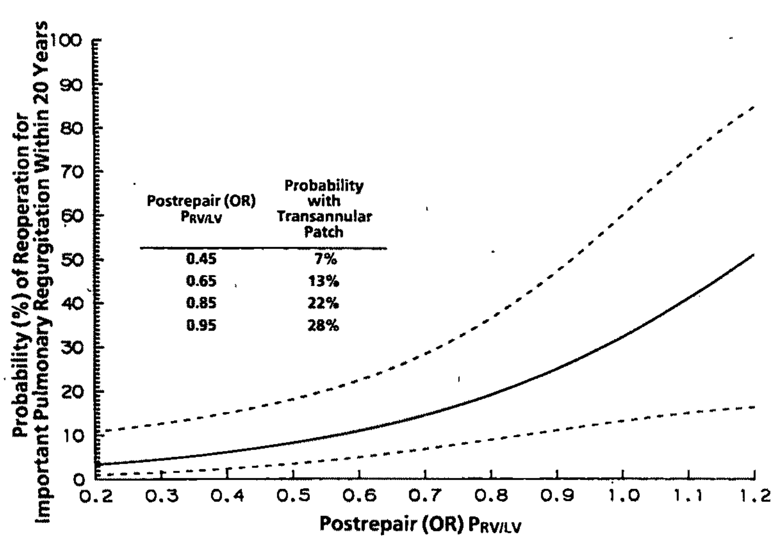


Fig 4. Specific solution of the multivariate equation relating the postrepair ratio between the peak pressure in the right ventricle and that in the left measured in the operating room (Postrepair (OR) $P_{RV/LV}$) *to the probability of reoperation for important pulmonary regurgitation within 20 years in patients in whom the repair had been performed using a transannular patch (see Appendix D for details).*

limits the inferences that can be drawn about the total patient population undergoing operation. The hemodynamic and exercise data available are compatible with the inferences derived from the current study.

Inferences as to the Detrimental Effect of Transannular Patching

It seems self-evident that pulmonary regurgitation and right ventricular volume overload follow the insertion of a transannular patch, even when the patch is not excessive in width. The time of greatest instantaneous hazard to survival must be when the volume overload is first evident after cardiopulmonary bypass and in the early hours thereafter, when only the functional myocardial reserve of the right ventricle is available for adaptation. The declining hospital mortality experienced in recent years among patients in whom a transannular patch has been used and the favorable experience with transannular patching in other institutions [10] are probably related to better protection of the myocardial reserves during operation. Thereafter, increase in right ventricular volume and change in wall characteristics aid in adaptation. However, the course of patients with isolated congenital pulmonary valve incompetence indicates that, although approximately 40 years of successful adaptation are usually allowed, the adaptive process ultimately fails in the face of continuing pulmonary regurgitation [11].

During the period of successful adaptation, as shown by this study, the patient's functional capacity is usually excellent, and hence, the probability of survival is not reduced, and reoperation is usually not indicated. Mild increase in right ventricular volume is part of the adaptive process [11]. The adaptive process is sufficiently robust that in most patients it is effective for at least 20 years, as seen in the present study and an earlier one by Fuster and colleagues [12]. However, objective exercise testing has

Table 4. Data on the 5 Patients Undergoing Reoperation (Insertion of Pulmonary Valve) for Pulmonary Regurgitation

Age at Repair	Operation Before Repair	Coexisting Anomalies	Postrepair (OR) $P_{RV/LV}$	Interval to Reoperation	$P_{RV/LV}$ Before Reoperation	RV Before Reoperation	Postreoperation (OR) $P_{RV/LV}$
19 mo	Right B-T shunt	Mild stenosis at LPA origin	0.42	8 mo	0.43	Dilatation grade 4/6	0.35
6 yr	Waterston shunt	Long stenosis in RPA	0.67	13 yr	. . .	. . .	0.63
4 yr	Palliative transannular patch	Severe RPA and LPA hypoplasia; large right collateral from innominate	0.68	9.5 yr	1.0 (same in PA)	Dilatation grade 4/6	(Inserted pulmonary valve was obstructive and removed)
7 yr	Waterston shunt	Obstruction of RPA	0.73	12 yr	0.52	Dilatation grade 6/6	0.41
8 yr	Potts' shunt	Stenosis at RPA origin; stenosis in mid-LPA	1.0	5 yr	0.8	Dilatation grade 5/6	1.05

B-T = Blalock-Taussig; LPA = left pulmonary artery; OR = operating room; PA = pulmonary artery; $P_{RV/LV}$ = ratio of peak pressure in right ventricle to that in left ventricle; RPA = right pulmonary artery; RV = right ventricle.

shown something less than normal capacity even during this period in nearly all patients in whom a transannular patch has been inserted [13].

As could be expected, there is some variability in the length of the period of successful adaptation, which in part probably accounts for a small proportion (12% in this study) of patients requiring reoperation within 20 years. The rising hazard function for reoperation in this group of patients (see Fig 3B) is consistent with the hypotheses developed here.

The success and duration of adaptation appear to be lessened by residual steriotic lesions beyond the transannular patch, including pulmonary vascular disease, according to the present study. This may relate to a possible increase in the regurgitant flow or an increase in right ventricular afterload. Because there is no certainty as to which mechanism is more responsible for the relations illustrated in Figure 4, there is no certainty that the presence of a pulmonary valve would change that relation.

When adaptation begins to fail, considerable benefit is provided by relieving the volume overload by orthotopic insertion of an allograft semilunar valve [14, 15], provided that this is not delayed until irreversible right ventricular damage has occurred.

Inferences as to Indications for Transannular Patching

Because clinical evidence of the detrimental effects of transannular patching is delayed for at least 20 years in most patients, efforts to preserve or substitute for the pulmonary valve should have at least as favorable an outcome. The relative risks of transannular patching, on the one hand, and of avoidance of transannular patching and preservation of the patient's pulmonary valve, albeit with a somewhat higher postrepair $P_{RV/LV}$, on the other, have not been clearly defined, but the strong unfavorable effect on outcome of increased resistance to right ventricular outflow must be taken into account [16]. Until these relative risks are specifically compared, transannular patching can be considered indicated when the predicted postrepair $P_{RV/LV}$ late postoperatively is greater than about 0.65. Also, the early and intermediate-term risks of insertion of an allograft valve have not as yet been determined to be as low as those of transannular patching. Consequently, until more information is available, the widespread use of this technique in the repair of tetralogy of Fallot with pulmonary stenosis is not indicated.

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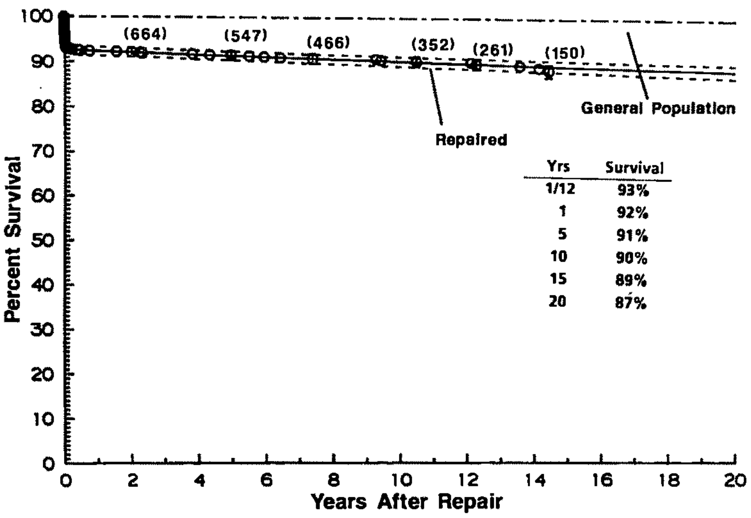
Appendix A. Analysis of Survival

The parameter estimates for the survivorship and hazard function were: Early phase: $\mu_1 = 0.07604$, $\delta = 0$, $\rho = 0.02600$, $v = 1$, and $m = -0.5420$; Constant phase: $\mu_2 = 0.0002557$ (Appendix Figs A-1, A-2).

Variables entered into the multivariate risk-factor analysis for death included *demographic and clinical variables*—age (years) at repair, body surface area (square meters) and weight (kilograms) at repair, sex, race, preoperative hematocrit; *previous operation variables*—previous operative procedure (yes/no, number, more than one), previous shunting procedure (classic Blalock-Taussig or Gore-Tex interposition shunt, single classic, right or left classic, Waterston, Potts', other), previous palliative direct relief of right ventricular outflow tract obstruction (Brock procedure [n = 13] or palliative transannular patch [n = 4]); *morphological variables*—anomalous origin of left anterior descending coronary artery from right coronary artery, patent ductus arteriosus, left superior vena cava, low-lying infundibular pulmonary stenosis, subarterial location of ventricular septal defect, large aortopulmonary collateral arteries, straddling tricuspid valve, multiple ventricular septal defects, dextrocardia (of the 5 patients,



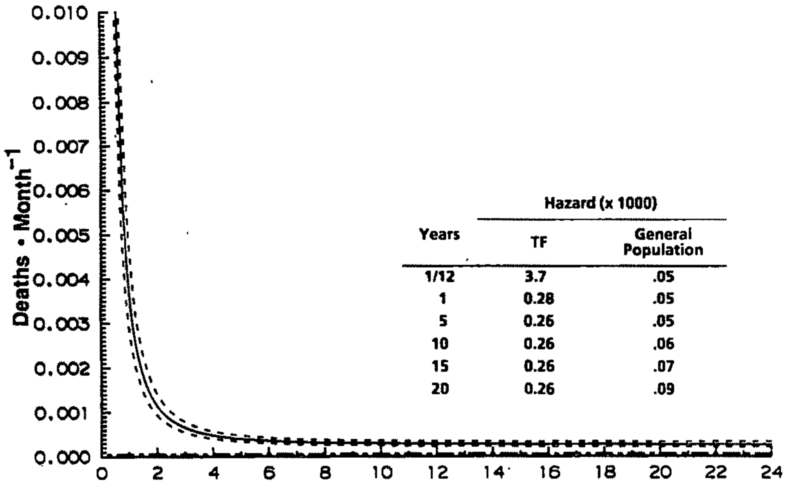
Yrs	Survival
1/12	93%
1	92%
5	91%
10	90%
15	89%
20	87%

Appendix Fig A-1. Survival after repair of tetralogy of Fallot with pulmonary stenosis (1967–May 1986) (n = 814). The circles represent a death, and are positioned along the horizontal axis according to the interval between the time of operation (time zero) and the death, and along the vertical axis actuarially. The vertical bars *represent the 70% confidence intervals (± 1 standard deviation). The* solid line *represents the parametric estimate of survival, and the* broken lines *enclose its 70% confidence intervals. The numbers in parentheses indicate the number of patients surviving and traced to the interval since operation. The* dash-dot-dash *line is the survival of an age-sex-race–matched general population (US Life Tables, 1976).*

2 had situs inversus totalis), severe tricuspid valve incompetence, partial anomalous pulmonary venous connection; *morphology of the pulmonary arteries variables*—left or right pulmonary artery stenoses, bifurcation stenosis, absence of unbranched hilar portion of one pulmonary artery, anomalous origin of a pulmonary artery from the ductus; *surgical variables*—atrial approach to ventricular septal defect, right ventriculotomy, type of repair (simple, transannular patch, or nonvalved extracardiac conduit, orthotopic valve, or valved extracardiac conduit), repair of left or right pulmonary artery stenosis, date of operation (years since January 1, 1967), use of total circulatory arrest, use of cardioplegia, global myocardial ischemic time using cardioplegia; and *postrepair (OR)* $P_{RV/LV}$.

Parameter estimates, coefficients, and p values for the equation representing the incremental risk factors for death resulting from the multivariate analysis in the hazard function domain were: Early phase: $\delta = 0$, $\rho = 0.02275$, $v = 1$, m = −0.4806, intercept = −6.729, *age at repair* = 0.006755 ± 0.0022 ($p = 0.002$), *body surface area* (natural logarithmic transformation) = −2.976 ± 0.57 ($p < 0.0001$), *hematocrit* = 3.554 ± 1.35 ($p = 0.008$), *more than one previous palliative operation* = 1.719 ± 0.45 ($p = 0.0001$), *multiple ventricular septal defects* = 1.404 ± 0.43 ($p = 0.001$), *dextrocardia* = 2.346 ± 1.04 ($p = 0.02$), *absence of unbranched hilar portion of one pulmonary artery* = 1.241 ± 0.38 ($p = 0.001$), *date of operation* = −0.09877 ± 0.034 ($p = 0.004$), *use of transannular patch* = 1.035 ± 0.30 ($p = 0.0006$), *postrepair (OR)* $P_{RV/LV}$ *(square transformation)* = 1.373 ± 0.48 ($p = 0.004$); Constant phase: intercept = −9.950, *age at repair* = 0.005298 ± 0.00113 ($p < 0.0001$), *previous Potts' anastomosis* = 1.269 ± 0.59 ($p = 0.03$), *previous palliative direct relief of right ventricular outflow tract obstruction* = 1.928 ± 0.60 ($p = 0.001$), *postrepair (OR)* $P_{RV/LV}$ *(squared transformation)* = 1.599 ± 0.79 ($p = 0.04$).

The nomogram in Figure 1 is a specific solution of the multivariate equation, and the values entered for each risk factor are as follows: *age*, 24 months; *body surface area*, 0.50 m^2; *hematocrit*, 0.5; *previous Potts' anastomosis, previous palliative direct relief of right ventricular outflow obstruction,*



	Hazard (x 1000)	
Years	TF	General Population
1/12	3.7	.05
1	0.28	.05
5	0.26	.05
10	0.26	.06
15	0.26	.07
20	0.26	.09

Appendix Fig A-2. Hazard function for death after repair of tetralogy of Fallot (TF) with pulmonary stenosis (1967–May 1986). The depiction is the same as in Appendix Figure A-1.

Appendix Table B-1. New York Heart Association Functional Class at Last Follow-up After Repair of Tetralogy of Fallot

	Category of Repair							
	Simple		Transannular Patch		Valved Conduit		Total	
Functional Class at Follow-up	No.	% of 478	No.	% of 201	No.	% of 17	No.	% of 696
I	459	96	196	98	14	82	669	96
II	15	3	4	2	3	18	22	3
III	4	1	1	0.5	0	0	5	1
IV	0	0	0	0	0	0	0	0
Subtotal	478	100	201	100	17	100	696	100
Dead	36		42		3		81	
Unknown	24		12		1		37	
Total	538		255		21		814	

more than one previous palliative operation, all No; *morphological risk factors* (see Table 1), all No; *date of operation,* 1986.

Appendix B. Functional Class Late Postoperatively

The Appendix Table B-1 contains the contingency table of New York Heart Association functional class at the time of the last follow-up. The ordinal logistic regression analysis was: intercept 1 = −3.669, intercept 2 = −5.389, *interval between repair and latest follow-up* (months) = 0.003734 ± 0.0032 (p = 0.24).

Appendix C. Reoperations

The indications for reoperation are shown in Appendix Table C-1.

Appendix Table C-1. Indications for Reoperation After Repair of Tetralogy of Fallot

Indication for Reoperation (n = 31) or Balloon Dilation (n = 1)	No. of Patients
Persistent or recurrent RVOTO[a]	17
Important pulmonary regurgitation	5
Residual or recurrent VSD	3
Large left-to-right shunt[b]	1
Valved conduit obstruction	2
Miscellaneous (one each)	4
Total	32

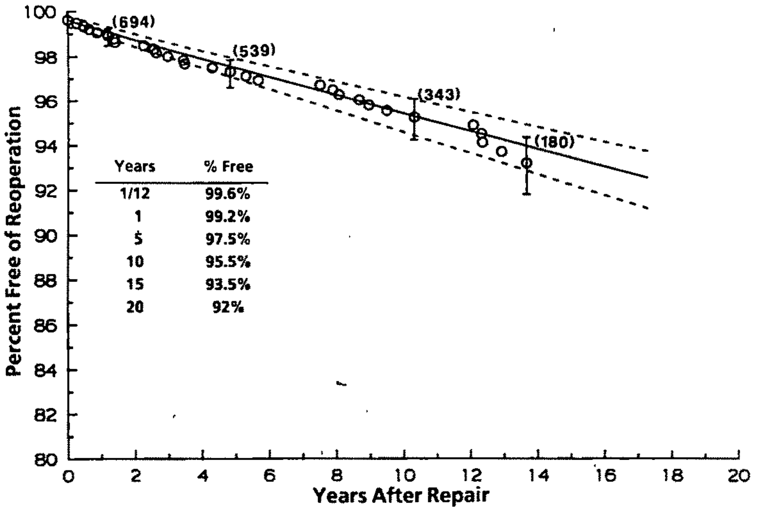
[a] In 13, this was the only abnormality at the time of reoperation; the obstruction was in the infundibulum in 4 patients, pulmonary annulus in 1, pulmonary artery bifurcation in 2, origin of the right and/or left pulmonary artery in 2, and multiple sites in 4. In 1 patient each, the RVOTO was associated with a small residual VSD, a small VSD and right ventricular aneurysm, a right ventricular aneurysm, and tricuspid incompetence. [b] Shunt was through a patent Blalock-Taussig shunt and a residual VSD.

RVOTO = right ventricular outflow tract obstruction; VSD = ventricular septal defect.

Parameter estimates for time-related depiction of reoperation of any kind (Appendix Fig C-1): Two hazard phases for reoperation of any kind were found. The shaping parameters for these were: Early phase: μ_1 = 0.003889, δ = 0, ρ = 0.03422, ν = 1, m = 0; Constant phase: μ_2 = 0.0003523.

Multivariate analysis of reoperation of any kind: The variables entered into the risk-factor analysis for reoperation of any kind included *demographic and clinical data*—age (years) at repair, sex, race, body surface area (square meters), hematocrit; *previous palliative procedures*—previous palliative procedures (yes/no, more than one, number), previous shunting procedures (classic, single classic, left classic, right classic, Waterston, Potts'), right ventricular outflow tract procedure (Brock or transannular patch); *morphological variables*—anomalous origin of left anterior descending coronary artery from right coronary artery, anomalous origin of one pulmonary artery, straddling tricuspid valve, patent ductus arteriosus, left superior vena cava, low-lying infundibular pulmonary stenosis, subarterial ventricular septal defect, large aortopulmonary collateral arteries, pulmonary artery morphology variables (left pulmonary artery stenosis, right pulmonary artery stenosis, bifurcation stenosis); *surgical variables*—atrial approach to ventricular septal defect, right ventriculotomy, type of repair (simple, transannular patch, valved conduit), repair of left pulmonary artery stenosis, repair of right pulmonary artery stenosis, date of operation (years since January 1967); and postrepair (OR) $P_{RV/LV}$ both as a numerical ratio and as a yes/no variable indicating a $P_{RV/LV}$ greater than or equal to 1.

Parameter estimates, coefficients, and p values for the multivariate risk-factor equation were: Early phase: δ = 0, ρ = 0.03487, ν = 1, m = 0, intercept = −7.448, *postrepair (OR) $P_{RV/LV}$* (squared transformation) = 3.970 ± 1.11 (p = 0.0003); Constant phase: intercept = −9.686, *body surface area* (natural logarithmic transformation) = −1.004 ± 0.47 (p = 0.03), *previous Potts' anastomosis* = 1.468 ± 0.78 (p = 0.06), *previous palliative direct relief of right ventricular outflow tract obstruction* (closed or open Brock) = 2.182 ± 0.76 (p =



Appendix Fig C-1. Actuarial and parametric depiction of percent freedom from reoperation of any kind after repair of tetralogy of Fallot with pulmonary stenosis (1967 to May 1986; $n = 814$; reoperations = 32). The circles, *actuarially positioned, represent individual patients undergoing reoperations and the* vertical bars, *the 70% confidence intervals. The patients at risk at certain actuarial time intervals are indicated by the numbers in parentheses. The* broken lines *represent the 70% confidence intervals around the* solid line, *which is the parametric estimate. Also shown are the parametric estimates at certain intervals since the original operation (see Appendix C for details).*

0.004), *valved extracardiac conduit* = 2.106 ± 0.74 (p = 0.005).

Appendix D. Reoperations for Important Pulmonary Incompetence

Parameter estimates for the time-related depiction of reoperation for important pulmonary regurgitation (the analysis was made only for the 255 patients in whom a transannular patch was used, as no reoperations for important pulmonary regurgitation were performed in patients in whom a transannular patch was not used): A single hazard-phase for reoperation for important pulmonary regurgitation was found: $\mu_3 = 0.004306$, $\tau = 134.3$, $\gamma = 1$, $\alpha = 0$, $\eta = 1$.

Variables entered into the multivariate analysis: The risk factors entered into the analysis were the same as in Appendix C except those describing repair of the pulmonary outflow tract were omitted.

Parameter estimates, coefficients, and p values for the multivariate risk factor equation were: Single phase: $\tau = 83.04$, $\gamma = 1$, $\alpha = 0$, $\eta = 1$, intercept = −6.819, *postrepair (OR)* $P_{RV/LV} = 3.040 \pm 1.85$ ($p = 0.1$).

This risk factor equation was solved for various postrepair (OR) $P_{RV/LV}$ values for Figure 4.

Coronary Arterial and Sinusal Anatomy in Hearts With a Common Arterial Trunk

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Eighty-four specimens of common arterial trunk were studied with special reference to the arrangement of the leaflets in relation to the atrioventricular valves, the origin of the coronary arteries in relation to the arterial sinuses, and the epicardial course of the coronary arteries. Fourteen normal hearts were used for comparison. In the hearts with common arterial trunk, the location and level of the coronary artery orifices (as well as the relationship of the truncal root to the area of fibrous continuity with the mitral valve) are different from those in normal hearts. In none of the hearts with common arterial trunk (particularly the 53 hearts with three leaflets in the truncal valve) did the appearance of the truncal valve approximate that of a normal aortic valve. Among the 22 hearts with four leaflets, there was a high incidence of coronary artery orifices in opposite sinuses (17/22 or 77.3%) and a low incidence of coronary artery orifices in adjacent sinuses (2/22 or 9.1%). These results suggest that the formation of the truncal valve is independent of the formation of the coronary orifices. Its leaflets are not predestined to become part of either the aortic valve or the pulmonary valve.

(Ann Thorac Surg 1989;48:792–7)

Several investigators [1–9] have demonstrated the variability in the formation of the leaflets and the origin of the coronary arteries in hearts with common arterial trunk. The morphogenesis of these arterial leaflets and sinuses remains the subject of controversy. Van Praagh [1–3] believes that the truncal valve is, in essence, the aortic valve; others [4, 10] disagree. To provide more circumstantial data concerning the morphogenesis of a common truncal valve, we investigated the arrangement of its leaflets in relation to the atrioventricular valves and the origin of the coronary arteries. In addition, we noted the anatomy of the epicardial course of the coronary arteries. This knowledge should prove of value in preventing injury to them during operation.

Material and Methods

We studied 84 heart specimens with common arterial trunk and compared them with 14 normal hearts. All were taken from the cardiopathological collections of The Hospital for Sick Children and Brompton Hospital in London. The patients had died between 1965 and 1988. Particular attention was directed toward the origin of the coronary arteries, their epicardial course, and their relationship to the semilunar leaflets and sinuses of the common arterial trunk. In the 14 normal hearts, we examined the aortic valve and the origin of the coronary arteries.

The width and depth of the valve leaflets were measured. The depth was considered the distance from the basal attachment of the leaflet to the free edge at its center. The width was determined by measuring the distance between the commissural attachments as a straight line. Hearts in which the largest leaflet was more than 1.5 times the width of the smallest were defined as showing an unbalanced arrangement of the leaflets.

In 1 heart, the truncal valve had been transplanted, and observation of the leaflets and the origin of the coronary arteries was not possible. In another heart, analysis of the coronary artery distribution proved impossible because of dissection during a previous study.

The χ^2 test was used for statistical analyses.

Results

Number of Truncal Leaflets

The truncal valve had two leaflets in 7 hearts (8.4%), three in 53 (63.9%), four in 22 (26.5%), and five in 1 heart (1.2%). A raphe through a leaflet was seen in 8 specimens: 6 hearts with three sinuses, 1 heart with four sinuses, and 1 heart with two sinuses (Table 1). An unbalanced arrangement of the leaflets was seen in 23 hearts with three leaflets (43%), 15 with four leaflets (68%), and 1 heart with two leaflets (14%). The incidence of unbalanced arrangement in hearts with four leaflets was significantly greater than that in hearts with two leaflets ($p < 0.05$) (Table 2).

Dysplastic Leaflets

Among the 53 hearts with three leaflets, 6 had dysplastic leaflets and 47, thick but almost normal leaflets (only one valve was judged to be completely normal). Among the 22 hearts with four leaflets, 14 had dysplastic leaflets and 8 were virtually normal. None of the hearts with two-leaflet valves had dysplastic features. Trunks guarded by four

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0003-4975/89/$3.50

Table 1. Summary of Valve Formation

Leaflets	Sinuses	Raphe	Total Hearts
3	3	6	53 (63.9)
4	4	1	22 (26.5)
2	2	1	7 (8.4)
5	5		1 (1.2)
Total	. . .	8	83

[a] Numbers in parentheses are percentages.

Table 3. Summary of Data on Leaflet Dysplasia

Leaflets	Dysplastic	Almost Normal	Total
3	6	47[a]	53
4	14	8	22
2	0	7[a]	7
5	1	0	1
Total	21	62	83

[a] $p < 0.01$ versus 4 leaflets.

leaflets had a significantly greater incidence of dysplastic leaflets than those with two or three leaflets (Table 3).

Origin of Coronary Arteries and Number of Sinuses

THREE LEAFLETS. Among the 53 hearts having three truncal leaflets, 8 hearts had a single coronary artery, and another 8 had a coronary artery orifice at the upper margin of a commissure (1 of these 8 hearts had a single coronary artery). In the remaining 38 hearts, two patterns of leaflet arrangement were discerned based on the relation of the truncal leaflets to the area of fibrous continuity with the leaflet of the mitral valve (Fig 1). In one group of 13 hearts, one truncal commissure was located more or less at the midregion of valvular continuity with the result that one leaflet was in an approximately posterior position. In the other group of 25 hearts, one commissure was at the extreme right end of the area of valvular continuity; thus, one leaflet was in an approximately anterior position.

When the coronary arteries of the hearts of these two groups were considered relative to their sinusal orientation, the left coronary artery arose from the left anterior sinus in 11 hearts and from the posterior sinus in 2 hearts in the first group. The right coronary artery arose from the right anterior sinus in 11 hearts, the left anterior sinus in 1 heart, and the posterior sinus in 1. In the second group, both coronary arteries arose from the left posterior sinus in 1 heart. In the remaining 24 hearts, the orifice of the right coronary artery was in the anterior sinus in 16 and the right posterior sinus in 8, and the left coronary artery arose from the left posterior sinus in 22 and the right posterior sinus in 2.

Table 2. Arrangement of Leaflets[a,b]

Leaflets	Balanced	Unbalanced
3	30	23 (43.4)
4	7	15 (68.2)
2	6	1 (14.3)[c]
5	0	1 (100)

[a] See Material and Methods section in text for definition of unbalanced arrangement. [b] Numbers in parentheses are percentages. [c] $p < 0.05$ versus 4 leaflets.

FOUR LEAFLETS. Of the 22 hearts with four leaflets, 3 had a coronary orifice at the upper margin of a commissure (13.6%). In another 2 hearts (9%), the coronary orifices arose from adjacent sinuses, and in the remaining 17 (77.3%), the coronary arteries arose from opposite sinuses (Fig 2). In this last group, the origin of the left coronary artery was mostly from a sinus forming a border of the area of truncal and mitral valve continuity (16 hearts); in 1 heart, it was from a remote sinus.

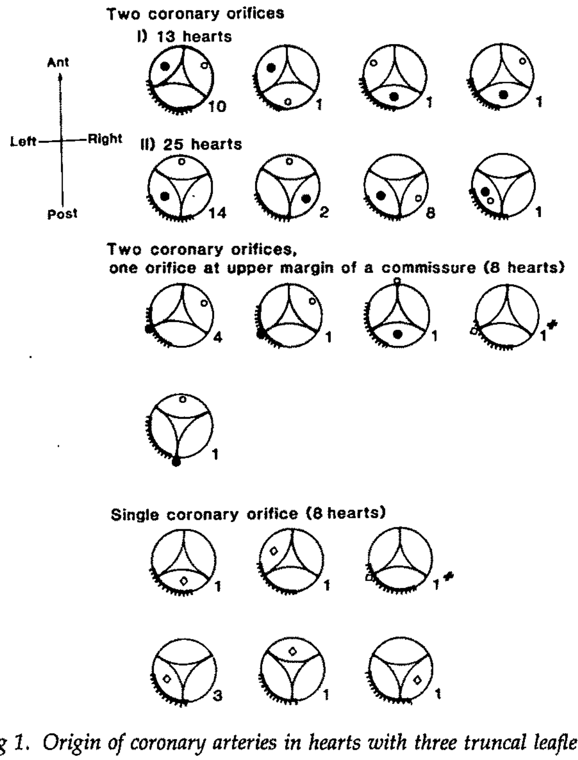

Fig 1. Origin of coronary arteries in hearts with three truncal leaflets. The right coronary artery is represented by an open circle *and the left coronary artery, a* closed circle. *(Top panel, I) One truncal commissure is located more or less at the midregion of valvular continuity, resulting in one leaflet being in a more or less posterior* (Post) *position. (Top panel, II) One commissure is at the extreme right end of the area of valvular continuity, with one leaflet being more or less in a directly anterior* (Ant) *position. (* ⟆ *= fibrous continuity between truncal valve and mitral valve; # = same heart; ◇ = single coronary artery.)*

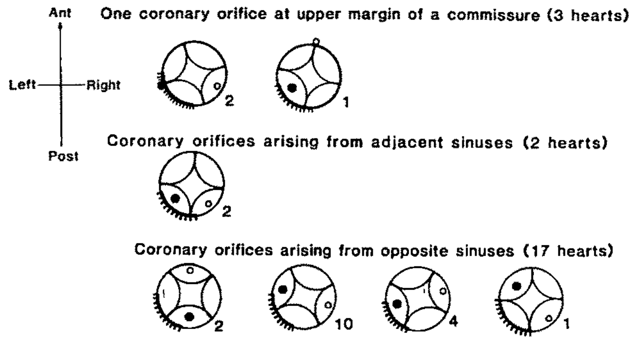
Ant
Left
Right
Post
One coronary orifice at upper margin of a commissure (3 hearts)
2
1
Coronary orifices arising from adjacent sinuses (2 hearts)
2
Coronary orifices arising from opposite sinuses (17 hearts)
2
10
4
1

Fig 2. Origin of coronary arteries in hearts with four truncal leaflets. Symbols and abbreviations are the same as in Figure 1.

TWO LEAFLETS. The arrangement of the orifices of the coronary arteries in the 7 hearts with two leaflets is shown in Figure 3. One heart had a single coronary artery and another, a coronary artery orifice at the upper margin of a commissure. In the remaining 5 hearts, the origin of the left coronary artery was from the anterior sinus in 1 heart with truncal and mitral valve continuity with the posterior sinuses and from the left lateral sinus in 4 hearts, the leaflet of the sinus itself being in continuity with the truncal valve.

Level of Coronary Orifices

The orifice of the left coronary artery was high (above the sinutubular bar) in 29 (39.2%) of 74 hearts (excluding the 9 with a single coronary artery), and the orifice of the right coronary artery had a high origin in 15 (20.3%). An orifice at the upper margin of a commissure was seen in another 11 hearts. The orifice of the left coronary artery was at the middle level of the sinus in 16 hearts (21.6%), and the orifice of the right coronary artery was at the same level in 16 hearts (21.6%). The orifice of the left coronary artery was lower than expected in 1 heart (1.4%) and that of the right coronary artery, in 2 hearts (2.7%). Among the 9 hearts with a single coronary artery, 5 showed a high origin (Table 4). In the 14 normal hearts, the origin of the orifice of the left coronary artery and of the right coronary artery was high in 2 hearts and 1 heart, respectively. The orifices of the other 25 left and right coronary arteries were at the midlevel of their respective sinuses.

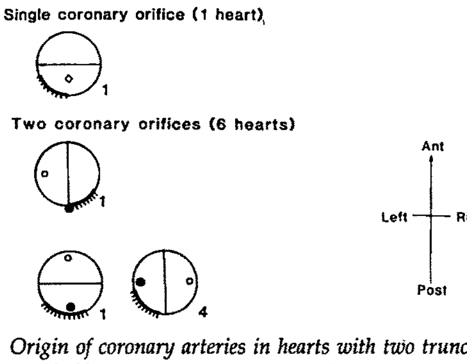
Single coronary orifice (1 heart)
1
Two coronary orifices (6 hearts)
1
1
4
Ant
Left
Right
Post

Fig 3. Origin of coronary arteries in hearts with two truncal leaflets. Symbols and abbreviations are the same as in Figure 1.

Table 4. Level of Coronary Ostia

Artery	High	Middle	Low[a]
LCA	29	16	29 (1)
RCA	15	16	43 (2)
Single	5	1	3 (0)

[a] Numbers in parentheses indicate the number of hearts in which the orifice was lower than expected.

LCA = left coronary artery; RCA = right coronary artery.

Location of Coronary Orifice

Of the 157 orifices of coronary arteries in hearts with common arterial trunk, 30 were located almost centrally within the sinus and 127 were at the margins of the sinus or at the upper margin of a commissure. In the 14 normal hearts, all 28 coronary artery orifices were located centrally in the sinus.

Ventricular Septal Defect and Mitral-Tricuspid-Truncal Valve Continuity

A ventricular septal defect in the superoanterior portion of the ventricular septum immediately beneath the truncal valve was present in all specimens with common arterial trunk. Fibrous continuity between the mitral and truncal valves was seen in all hearts except 1. In this heart, the ventriculoinfundibular fold completely separated the truncal valve from both the mitral and the tricuspid valves. In 76 hearts, the defect had a muscular posteroinferior rim separating the mitral and tricuspid valves. In 8 (9.5%) of the 84 hearts, there was fibrous continuity between the truncal valve and both the mitral and tricuspid valves, so that the ventricular septal defect was perimembranous.

Only 8 (21.1%) of the 38 hearts with three leaflets and two coronary arteries showed continuity between the anterior leaflet of the mitral valve and more than half of the extent of the noncoronary leaflet of the truncal valve. Of the other 30 hearts, 29 (76.3%) had fibrous continuity between the left coronary leaflet and the mitral valve, and 1 heart had continuity between the right coronary leaflet and the mitral valve (Fig 4). All but 1 of the normal hearts showed valvular continuity between more than half of the extent of the noncoronary aortic leaflet and the mitral valve. One heart had continuity between the mitral valve and more than half of the extent of the left coronary aortic leaflet.

Epicardial Distribution of Coronary Arteries

The right coronary artery was dominant in 64 (85.3%) of 75 hearts (excluding hearts with a single coronary artery), and the left coronary artery was dominant in 10 (13.3%). A balanced arrangement was found in 1 heart (1.3%). The artery to the sinus node arose from the left coronary artery in 15 (22.1%) of 68 hearts (excluding those with a single ostium and 8 with an undetectable sinus node artery, 1 heart being in both categories) and from the right coronary artery in 53 hearts (77.9%). Among the 9 hearts with a single coronary artery, the sinus node artery arose from

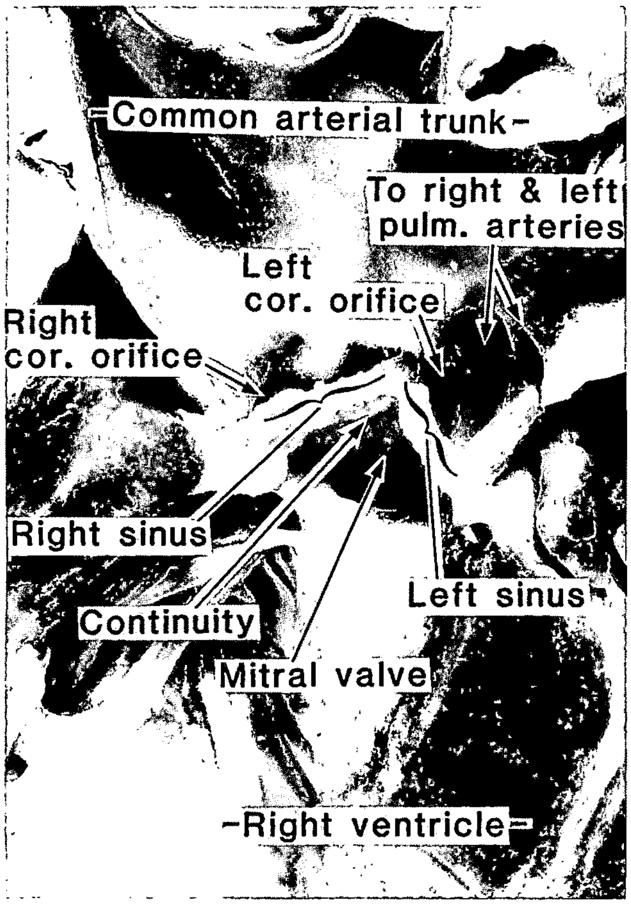

Fig 4. Continuity in 1 heart specimen between the right coronary leaflet and the mitral valve as seen through the ventricular septal defect.

its circumflex branch in 2 and from the right coronary artery in 6. The origin of the sinus node artery could not be detected in 1 heart.

The circumflex coronary artery was absent in 2 hearts with a single coronary artery and in 1 of the other hearts. It was hypoplastic in 5 hearts and originated from the right coronary artery in another 3 hearts.

The left anterior descending coronary artery arose from the right coronary artery in 2 hearts (1 with a single coronary artery). The circumflex and anterior descending coronary arteries had separate orifices from the same truncal sinus in 1 heart; dual orifices for the right coronary artery and its infundibular branch were seen in another heart. Atresia of the left coronary artery was seen in 1 heart, and the left main trunk was absent in another (Table 5).

Associated Cardiac Abnormalities

The associated cardiac abnormalities are shown in Table 6.

Comment

The variability in the formation of the leaflets of the common arterial valve and in the origin of the coronary arteries in hearts with common arterial trunk is well established [1–8, 10]. In contrast, the arrangement of the leaflets in relation to the atrioventricular valves and the origin of the coronary arteries in relation to the truncal sinuses are not as well known. The precise anatomy of the epicardial course of the coronary arteries is also less well known. Apart from the intrinsic value of such knowledge for the surgeon, it can throw light on the continuing controversy concerning the morphogenesis of hearts with common arterial trunk.

Although others [4, 10] do not concur, Van Praagh [1–3] maintains that the truncal valve is basically the aortic

Table 5. Summary of Data on Coronary Artery Anatomy[a]

Anatomy	No. of Hearts
LCA dominance	10/75 (13.3%)
RCA dominance	64/75 (85.3%)
Balanced arrangement	1/75 (1.3%)
Sinus node artery from LCA	15/68 (22.1%)
Sinus node artery from RCA	53/68 (77.9%)
Single coronary artery	9/84 (10.7%)
LCx artery from RCA	3
LAD from RCA	2 (1 with single coronary artery)
Absent LCx	3 (2 with single coronary artery)
Hypoplastic LCx	5
Absent left main trunk	1
Dual orifice of LCA	1
Dual orifice of RCA	1
Common sinus origin of LCA and RCA	1
Atresia of LCA orifice	1

[a] Numbers in parentheses are percentages.

LAD = left anterior descending coronary artery; LCA = left coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery.

Table 6. Associated Cardiac Abnormalities[a]

Abnormality	No. of Hearts
Interruption of aortic arch	11 (13.1)
Coarctation of aortic arch	2 (2.4)
Right aortic arch	10 (11.9)
Anomalous origin of subclavian artery	8 (9.5)
Absent superior caval vein	1 (1.2)
Atrial septal defect	22 (26.2)
Patent left superior caval vein	5 (6.0)
Patent arterial duct (excluding interruption)	10 (11.9)
Partial anomalous pulmonary venous connection	1 (1.2)
Multiple ventricular septal defects	1 (1.2)

[a] Numbers in parentheses are percentages.

valve. He believes that this explains why it is usually tricuspid and why, when some pulmonary leaflet tissue persists (a condition made possible by postulated absence of the aortopulmonary septum at the level of the arterial valves), the truncal valve can be quadricuspid. If this were the case, the truncal valve with three leaflets should have the basic formation of an aortic valve, and its sinuses and their relationship to the atrioventricular valves should be comparable with those found in the normal heart.

In fact, however, in none of our hearts (particularly those with three leaflets) did the appearance of the truncal valve approximate that found in the normal aortic valve. Furthermore, the level of the coronary artery orifices and their locations within the sinuses of the common trunk were markedly different from those in the normal aorta. The majority of the orifices were situated at a high level or close to the margins of the sinuses. Only a few were found at a relatively normal location within the middle of the sinuses. Even in those hearts, the orifice of the other coronary artery was usually abnormally located. Taken together, our findings endorse strongly the conclusions of Crupi and colleagues [8], namely, that the structure of the truncal root with three leaflets and sinuses cannot be interpreted to indicate that it represents the aortic root.

As for the truncal valve with four leaflets, only 2 hearts (9.1%) had coronary orifices in adjacent sinuses. In all the others (77.3%) (excluding 3 hearts with the orifice at the upper margin of a commissure), the coronary arteries arose from opposite sinuses. If the four leaflets of the truncal valve originated from the main truncal ridges as Van Praagh [1–3] has suggested, two of the four leaflets ought to develop into aortic leaflets and the other two, into pulmonary leaflets. The coronary arteries would then be expected to arise from adjacent sinuses. In contrast, our results show a high incidence of coronary orifices located in opposite sinuses and a low incidence of coronary orifices located in adjacent sinuses as seen in the aortic valve. This suggests that the formation of the truncal valve is independent of the formation of the coronary orifices, and that the four leaflets are never destined to become part of either the aortic or the pulmonary valve.

Among our heart specimens, those with four leaflets showed a higher incidence of dysplasia and unbalanced formation of the leaflets than did those with three or two leaflets. We also had one dysplastic valve with five leaflets. These findings suggest to us that most truncal valves are derived from four or more anlagen, and that fusion of leaflets and sinuses occurs during development to produce the variants with three or two sinuses and leaflets. In this respect, raphes were seen especially on those hearts with three leaflets, thus suggesting an incomplete division into four sinuses.

The high incidence of a single coronary artery and the tendency for the left coronary artery to arise from a position toward the posterior wall of the trunk have been previously reported [1–8]. In addition to confirming these findings, we noted additional variations in the distribution of the coronary arteries, variations with important surgical implications. Origin of the left circumflex coronary artery from the right coronary artery is known to be a frequent variation in hearts with complete transposition [11], though not described in hearts with common arterial trunk. We found it in 3 hearts (3.6%). Absence of the left circumflex artery is very rare in normal hearts [12]. We found this anomaly in 3 (3.6%) hearts. Origin of both coronary arteries from the same sinus is also rare in normal hearts [13], but this was found in 1 of our hearts. Origin of the left anterior descending coronary artery from the right coronary artery, recognized as a major anomaly in tetralogy of Fallot (4.2%) [14], was seen in 2 (2.4%) of our hearts with common arterial trunk.

The incidence of left coronary artery dominance, however, seems to be about the same as in normal hearts (13.3% and 10% to 12%, respectively [15]). On the other hand, origin of the sinus node artery from the left coronary artery was seen less frequently than in normal hearts (22.1% and 39%, respectively [16]).

In our observations of these 84 hearts with common arterial trunk, we found 3 hearts in which the coronary arteries had been injured by surgical procedures. The course of these vessels, if anomalous, should be recognized before operation [2]. Our study indicates that this should be anticipated in a substantial number of patients.

During the course of this study, Atsuko Suzuki was a visiting fellow from the National Cardiovascular Center in Osaka, Japan.

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Preservation of Pancreatic Beta Cell Function With Pulsatile Cardiopulmonary Bypass

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Pancreatic islet cell function and tissue metabolism were studied during and after cardiopulmonary bypass in 38 patients undergoing an open heart operation. Twenty patients were operated on with pulsatile cardiopulmonary bypass (group I) and 18, with nonpulsatile cardiopulmonary bypass (group II). Hyperglycemia was observed during and early after operation in both groups. In group I during cardiopulmonary bypass, the immunoreactive insulin and c-peptide levels and the insulin to glucagon molar ratio increased significantly compared with the preoperative values, but in group II, these variables did not alter significantly. An hour postoperatively, the immunoreactive insulin (71 ± 34 μIU/mL) and c-peptide (8.3 ± 3.0 ng/mL) levels and the insulin to glucagon molar ratio (11.0 ± 5.2) in group I were significantly higher than those in group II (immunoreactive insulin, 29 ± 20 μIU/mL; c-peptide, 4.8 ± 1.8 ng/mL; insulin to glucagon molar ratio, 3.4 ± 2.6). The blood lactate level in group I (41 ± 22 mg/dL) was significantly lower than that in group II (78 ± 30 mg/dL) an hour postoperatively. In conclusion, pulsatile cardiopulmonary bypass is quite effective in preserving pancreatic beta cell function and tissue metabolism during and early after open heart procedures.

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In a critically ill patient, such as one with severe trauma, surgical trauma, or burns, plasma insulin levels might remain within the normal fasting levels despite coexisting hyperglycemia and hyperglucagonemia due to disorders of pancreatic islet cell, ie, alpha and beta cell, function. This results in carbohydrate metabolic disturbances accompanied by lowering of the insulin to glucagon (I/G) ratio [1–4].

Although changes in carbohydrate and tissue metabolism during and after cardiopulmonary bypass (CPB) for open heart procedures have been demonstrated by some authors [5–8], the effects of pulsatile CPB on pancreatic islet cell function have not been specifically investigated to date [9–11]. The purpose of this study was to examine the influence of pulsatile CPB on insulin-glucagon relationships, especially pancreatic beta cell function and tissue metabolism, during and early after open heart operations.

Material and Methods

Thirty-eight consecutive, nondiabetic patients who underwent an elective cardiac operation were entered into this study. Twenty-one had coronary artery bypass grafting and 17, a valve operation. The patients were divided into two groups, based on the type of CPB. Twenty patients had pulsatile CPB using the AVCO pulsatile bypass system (model 20; AVCO Medical Products) (group I), and 18 had nonpulsatile CPB (group II). The groups were comparable in terms of age, sex, and type of cardiac disease (Table 1).

Cardiopulmonary bypass was performed using a William Harvey H-1500 bubble oxygenator with a double-roller pump. Lactated Ringer's solution was used as the glucose-free priming diluent of the system, and the following agents were added to the solution: 7% sodium bicarbonate (40 to 60 mL), heparin (5,000 U/1,000 mL), 10% calcium chloride (5 mL/200 mL of acid-citrate-dextrose blood), and acid-citrate-dextrose blood.

Table 2 provides additional perfusion information. There were no significant differences between the two groups except for pulse pressure, which was significantly greater in group I than in group II (Fig 1).

Blood Collection and Measurements

Serial blood samples were obtained from the radial or femoral artery in all patients for measurements early in the morning of the operative day, one hour after the beginning of total CPB, and 1, 5, 24, and 48 hours postoperatively. Blood glucose level was determined by the oxygen sensor method. Plasma levels of immunoreactive insulin (IRI) were determined by the radioimmunoassay solid-phase method [12] (normal values in our laboratory range from 7 to 24 μIU/mL). Plasma levels of c-peptide [13] and immunoreactive glucagon [14] were analyzed by the radioimmunoassay double-antibody technique (normal values range from 1.5 to 3.5 ng/mL for c-peptide and from 70 to 160 pg/mL for glucagon). Plasma lactate levels were determined by the ultraviolet technique [15] (normal values range from 9 to 16 mg/dL).

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Table 1. Group Profiles

Variable	Group I (Pulsatile) (n = 20)	Group II (Nonpulsatile) (n = 18)
Age (yr)	17–65 (mean, 46.1)	22–64 (mean, 45.5)
Sex		
Male	12	12
Female	8	6
Cardiac disease		
Ischemic	10	11
Valvular	10	7

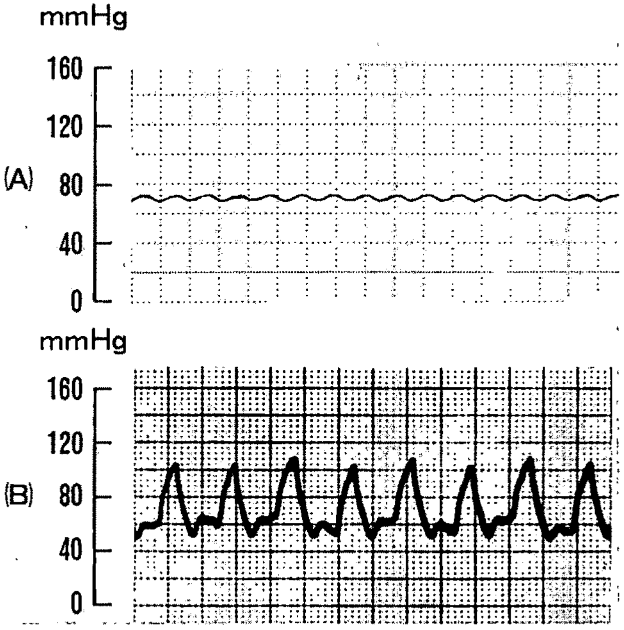

Fig 1. Pressure tracings during (A) nonpulsatile cardiopulmonary bypass (group II) and (B) pulsatile cardiopulmonary bypass (group I).

Statistical Analysis

All data are shown as the mean ± the standard error. Significance was determined by the Student paired and unpaired t tests. A p value of less than 0.05 was considered significant.

Results

During CPB, blood glucose and IRI levels in group I were significantly elevated compared with the preoperative values (Table 3). The c-peptide levels were significantly higher in both groups compared with the preoperative values. The immunoreactive glucagon level and the I/G molar ratio showed no significant change in either group. In group II, the lactate level was significantly elevated compared with the preoperative value. The hematocrit was significantly lower in both groups compared with the preoperative values.

During the postoperative period, blood glucose levels in both groups were significantly greater one and five hours postoperatively compared with the preoperative values (see Table 3). The IRI level, c-peptide level, and I/G molar ratio in group I were also significantly higher one and five hours postoperatively compared with the preoperative values, but in group II, these variables showed no significant changes. Lactate levels in both groups were significantly elevated one and five hours postoperatively compared with the preoperative values. The hematocrit had recovered to the preoperative values one hour postoperatively in both groups.

One hour postoperatively, the IRI level, c-peptide level, and I/G molar ratio in group I were significantly higher than those in group II (see Table 3). The lactate level in group I was significantly lower than that in group II. Moreover, 6 (42.9%) of 14 patients in group II had high lactate levels of more than 80 mg/dL. In contrast, only 1 (7.1%) of 14 patients in group I showed a lactate level of more than 80 mg/dL (Fig 2). This difference between the two groups was significant ($p < 0.001$). Five hours postoperatively, the IRI level, c-peptide level, and I/G molar ratio in group I were significantly higher than those in group II, but there was no significant difference between the lactate levels in the two groups.

Comment

The controversy over the clinical use of pulsatile versus nonpulsatile CPB in open heart surgery has continued without immediate prospect of resolution. Some authors have reported that pulsatile CPB offers many advantages compared with nonpulsatile CPB, including lower vascular resistance [7, 16, 17], lower blood lactate level [7, 8, 16], increased urine volume [8], lower plasma concentration of

Table 2. Perfusion Data[a]

Group	Total CPB Time (min)	Flow Rate (L/min/m²)	Mean Pressure (mm Hg)	Pulse Pressure (mm Hg)	Body Temperature (°C)
Group I, pulsatile (n = 20)	99.5 ± 28.0	2.6 ± 0.1	62.5 ± 9.0	37.1 ± 6.7[b]	26.4 ± 1.6
Group II, nonpulsatile (n = 18)	84.5 ± 22.5	2.5 ± 0.1	57.0 ± 20.8	15.2 ± 6.3	26.3 ± 2.1

[a] Data are shown as the mean ± the standard error. [b] Significance: $p < 0.001$ versus group II.

CPB = cardiopulmonary bypass.

Table 3. Changes in the Variables Studied[a]

		Sampling Times					
				Postop (h)			
Variable	Group	Preop	Cardiopulmonary Bypass	1	5	24	48
Blood glucose (mg/dL)	I	83 ± 18	206 ± 44[b]	253 ± 51[c]	235 ± 52[c]	125 ± 46	115 ± 27
	II	87 ± 27	254 ± 92[c]	281 ± 67[c]	271 ± 60[c]	208 ± 72[b]	149 ± 25
Immunoreactive insulin (μIU/mL)	I	16 ± 9	72 ± 39[c]	71 ± 34[b,d]	89 ± 36[c,d]	72 ± 42	64 ± 35
	II	13 ± 5	24 ± 15	29 ± 20	40 ± 27	31 ± 23	27 ± 18
C-peptide (ng/mL)	I	1.7 ± 1.0	6.8 ± 1.7[d,e]	8.3 ± 3.0[f,g]	7.4 ± 2.5[d,f]	3.3 ± 1.6	4.3 ± 1.6
	II	1.6 ± 0.7	4.4 ± 1.2[h]	4.8 ± 1.8	4.1 ± 1.7	3.2 ± 1.3	3.6 ± 1.2
Immunoreactive glucagon (pg/mL)	I	98 ± 34	150 ± 81	148 ± 73	202 ± 101	225 ± 88	220 ± 91
	II	107 ± 45	200 ± 96	197 ± 88	221 ± 79	175 ± 80	156 ± 66
Insulin to glucagon molar ratio	I	3.8 ± 1.4	11.2 ± 6.5	11.0 ± 5.2[e,g]	10.2 ± 5.2[d,e]	7.3 ± 4.0	6.3 ± 3.3
	II	3.1 ± 1.0	2.9 ± 1.5	3.4 ± 2.6	4.2 ± 2.5	3.7 ± 2.7	4.2 ± 2.5
Lactate (mg/dL)	I	18 ± 3	37 ± 20	41 ± 22[d,e]	38 ± 14[b]	23 ± 16	16 ± 8
	II	15 ± 4	59 ± 29[e]	78 ± 30[e]	50 ± 18[c]	17 ± 9	18 ± 10
Hematocrit (%)	I	40.0 ± 4.0	21.4 ± 3.3[c]	39.3 ± 2.5	36.8 ± 2.9	37.0 ± 3.3	36.6 ± 3.8
	II	37.7 ± 4.0	22.7 ± 3.8[c]	37.7 ± 4.2	37.5 ± 2.5	37.6 ± 2.7	37.1 ± 3.2

[a] Data are shown as the mean ± the standard error. [b] Significance: $p < 0.005$ versus preoperative value. [c] Significance: $p < 0.001$ versus preoperative value. [d] Significance: $p < 0.05$ versus group II. [e] Significance: $p < 0.01$ versus preoperative value. [f] Significance: $p < 0.02$ versus preoperative value. [g] Significance: $p < 0.01$ versus group II. [h] Significance: $p < 0.05$ versus preoperative value.

the vasoconstricting agents angiotensin II [9, 18] and vasopressin and other stress hormones [10], better cellular oxygenation [17], and improved organ function [17]. On the other hand, other authors [11, 19, 20] have failed to show major advantages of pulsatile CPB.

The stress response to hypothermic nonpulsatile bypass in humans has been shown to produce hyperglycemia as well as suppression of insulin secretion, which is called "surgical diabetes," during and after CPB [5, 6, 21]. This glucose intolerance results in a low serum potassium level because of intracellular displacement of potassium, which could increase the possibility of cardiac arrhythmias and a diminished cardiac performance after anoxic arrest secondary to depressed uptake of glucose into the cardiac muscle [6]. The explanation for this suppression of insulin secretion during open heart procedures may be related to the reduction in metabolic activity of the pancreas caused by hypothermia [6], reduction in pancreatic blood flow [22], and release of stress hormones previously observed during and after nonpulsatile CPB [10, 18, 23–25].

It is well known that insulin and glucagon have a competitive antagonism on carbohydrate metabolism [1, 25, 26]. Moreover, it has been reported that the I/G molar ratio, not the absolute concentration of insulin and glucagon, is the important signal predicting protein anabolism or catabolism in surgical or trauma patients [1, 4, 25]. In normal subjects, the ingestion of exogenous amino acids, such as protein meal, induces a rise in the ratio of insulin to glucagon from the normal fasting level I/G molar ratio of 3.9 to a peak level of 6.3 because of the normal pancreatic beta cell function [1]. This increase in the ratio would favor protein synthesis, that is, protein anabolism. Moreover, hyperglycemia following an exogenous glucose load could induce the promotion of insulin secretion accompanied by the suppression of glucagon release, resulting in a rise in the I/G molar ratio to 15.0 [1, 27].

In contrast, in surgical patients or those with severe trauma or burns, because the pancreatic alpha cell function is activated and thus the beta cell function is adversely suppressed [2, 3], the plasma glucagon level rises significantly, whereas the plasma insulin level remains within normal limits despite the presence of hyperglycemia; consequently, the I/G molar ratio falls to less than 2.0 [2–4]. This decrease in the I/G molar ratio might cause increased hepatic glucose production from glycogen and available amino acids, and consequently a negative nitrogen balance, that is, protein catabolism [1, 4].

In the present study, it was observed that plasma IRI and c-peptide levels were elevated in response to hyperglycemia without a significant change in the glucagon level during CPB and one and five hours after CPB in the pulsatile CPB group (group I), whereas no significant elevations of these substances were observed in the nonpulsatile CPB group (group II) despite elevations of blood glucose levels. The I/G molar ratio correlatively increased one and five hours postoperatively only in group I. Furthermore, the plasma IRI level, c-peptide level, and I/G molar ratio in group I were significantly higher than those in group II one and five hours postoperatively.

These findings suggest that pulsatile CPB definitely maintains the responses of insulin secretion to hyperglycemia during and after CPB even in moderate hypothermia and thus could preserve protein anabolism in the early postoperative period. It has been reported that circulating levels of c-peptide well reflect pancreatic beta

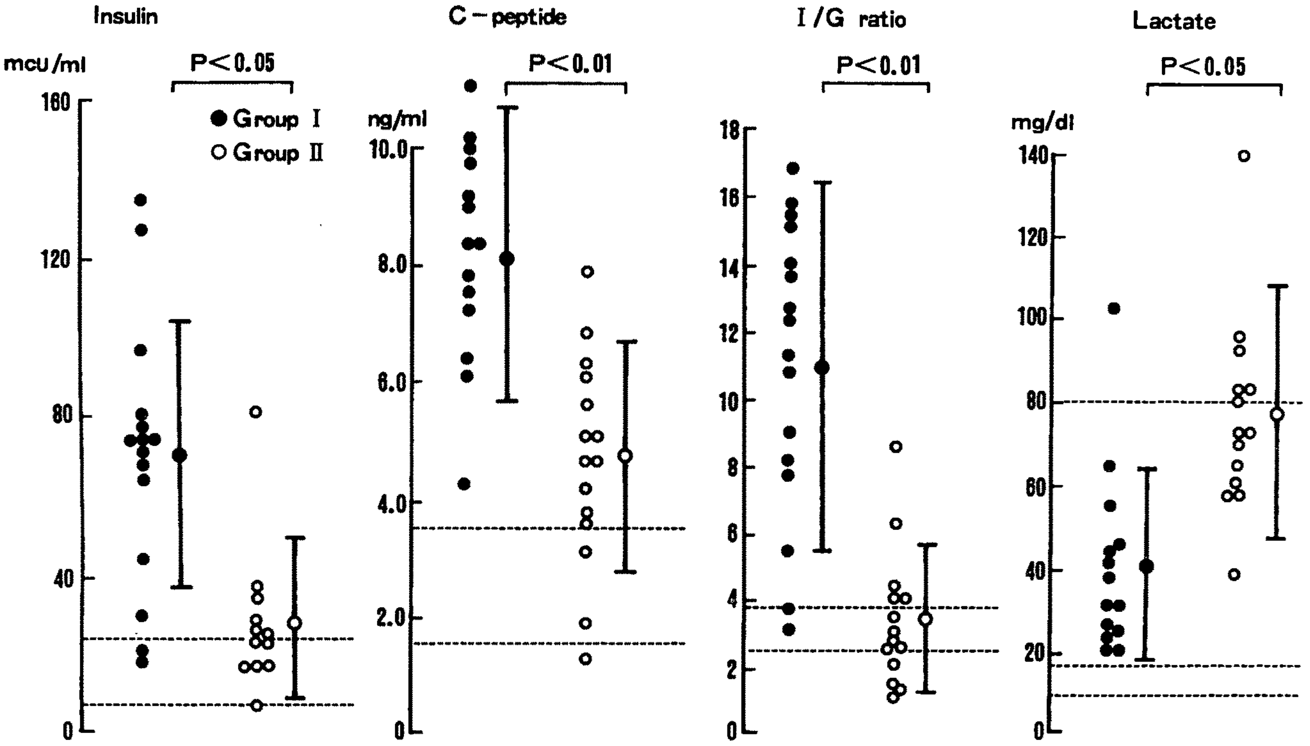

Fig 2. Effect of pulsatile cardiopulmonary bypass (CPB) (group I) versus nonpulsatile CPB (group II) on plasma insulin levels, c-peptide levels, the insulin to glucagon molar ratio (I/G ratio), and lactate levels one hour postoperatively. The levels of insulin and c-peptide and the I/G molar ratio in group I were significantly higher than those in group II. However, the lactate level in group I was significantly lower than that in group II.

cell secretory capacity in a patient with disorders of glucose homeostasis [11]. Therefore, it was demonstrated in this study that pulsatile perfusion probably maintains pancreatic beta cell secretory activity during and early after an open heart operation, although there was no evidence of substantial activation of the pancreatic alpha cell function during and after operation in either group. Several investigators [7–9] have observed that the increase in blood lactate levels that occurs during conventional nonpulsatile CPB is actually prevented by the use of pulsatile flow. In contrast, others [11, 20] have failed to find any difference in oxygen consumption and lactate levels between pulsatile CPB and nonpulsatile CPB groups.

This study demonstrated that lactate levels showed peak high values an hour postoperatively in both groups. This peak value in the pulsatile CPB group was significantly lower than that in the nonpulsatile CPB group. It has been reported that glucagon stimulates hepatic lactate production [25]. However, as the glucagon level showed no significant elevation in this study, the major cause of the increase in lactate levels during and after CPB is thought to be tissue hypoxia due to low perfusion. Perez and co-workers [28] suggested that patients who are in shock and have a high lactate level (more than 80 mg/dL) have a grave prognosis with high mortality.

It was shown in the present study that an hour postoperatively, pulsatile CPB resulted in a significantly lower incidence of lactate levels of greater than 80 mg/dL, the critical level, compared with nonpulsatile CPB. These findings strongly suggest that pulsatile CPB offers better tissue perfusion than nonpulsatile CPB during and early after operation. These data indicate that more physiological metabolic variables are preserved with pulsatile CPB than with nonpulsatile CPB during and early after an open heart procedure.

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New Outlook on Pericardial Substitution After Open Heart Operations

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The difficulties of reoperation owing to adhesions are well known. Clinical attempts to solve this problem using synthetic materials or glutaraldehyde-fixed pericardial xenograft have been less than satisfactory. Although experimental animal results have been good, they have not considered the influence of cardiopulmonary bypass (CPB) on adhesion formation. This study addressed the influence of CPB on the formation of adhesions and evaluated biodegradable polyglycolic acid as a material to reduce adhesions and as a pericardial substitute. Forty-five weanling sheep received implants of pericardial xenograft and polyglycolic acid with and without CPB. The pericardial xenograft showed no adhesions when implanted without CPB, but severe adhesions formed and a thick fibrinous layer covered the heart when CPB was used, making identification of coronary arteries at reoperation very difficult. The polyglycolic acid mesh implanted without CPB was absorbed and replaced with newly formed host collagen. With CPB, the polyglycolic acid was more rapidly absorbed, and a thinner layer of host collagen formed. Therefore, future animal studies must include CPB. In agreement with reported clinical results, glutaraldehyde-fixed pericardial tissue implanted with CPB resulted in severe epicardial reaction and therefore is not an ideal pericardial substitute.

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Reoperation after open heart operations is becoming more common. In 1987, about 300,000 coronary artery graft bypasses were performed in the United States, with a reoperation rate of 10% to 20% per year. Morbidity and mortality have increased owing to severe adhesions formed between the heart and the sternum [1]. Reoperation after open heart operations is also made more difficult when the native pericardium cannot be approximated and is left open because of the risk of pressure on the aortocoronary grafts. In an edematous heart, closure of the pericardium can also lower the performance of the heart [2, 3]. Prevention of adhesions through pericardial substitution has therefore become a matter for investigation.

Many attempts have been made to solve the problem of adhesions by using different types of pericardial substitutes [4, 5]. These have included silicone membranes [6], polyurethane [7], fascia lata [8, 9], Gore-Tex polytetrafluoroethylene (PTFE) patches [7, 10, 11], bovine or porcine pericardium xenografts (PXs) treated with glutaraldehyde [12–15], siliconized Dacron [16, 17], and dura mater [18]. All experimental trials of these pericardial substitutes were performed in dogs or small laboratory animals using regular thoracotomy or sternotomy and without cardiopulmonary bypass (CPB).

Most pericardial substitute materials have given good results in these experimental animal studies, but clinical results, especially for the long term, have not been satisfactory [19–21]. Gallo and associates [13] reported that even if the pericardial substitutes were not adherent to the heart at the time of reoperation, a thick fibrinous layer subsequently covered the epicardium, making it nearly impossible to perform a second coronary bypass procedure.

Experimental results using animals have been far superior to the results of human clinical trials, which have demonstrated varying degrees of success. We believe that the differences noted between animals and humans may arise from differences in protocols. The experimental animal protocols have not used a CPB technique common to human experience. There are some reports on the damaging effects of CPB on the pericardium and the epicardium in humans, including impairment of the fibrinolytic activity of the pericardium [22, 23]. Epicardial irritation and petechiae can also cause severe tissue reactions, which may precipitate severe adhesions. Some investigators have further suggested that the fibrinolytic activity of the pericardium may prevent fibrin or blood clot formation, which can become the base for adhesions, and that CPB can lower or abolish this activity. Therefore, we believe that comparison of animal results without CPB to human clinical experience with CPB may be a meaningless exercise.

The purpose of this investigation was threefold. First, we wanted to determine the feasibility of using an absorbable material as a pericardial substitute. This included investigation into absorption rate and the nature of the tissue reaction to the absorbable material. Second, we wanted to compare a biological material (bovine pericar-

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Table 1. Pericardial Substitutes Implanted

Group No.	Type of Pericardial Substitute	CPB Used	No. of Sheep	Surgical Procedure
1	Glutaraldhyde-fixed bovine pericardium from mixed sources[a]	No	9	Thoracotomy
2	Dexon (PGA) and Maxon combinations	No	18	Thoracotomy
3	Glutaraldehyde-fixed bovine pericardium (xenograft Periguard[b])	Yes	12	Sternotomy
4	PGA[c] with and without Dacron mesh	Yes	6	Sternotomy

[a] Periguard, Ionescu-Shiley, and home-made. [b] Bio Vascular, St Paul, MN. [c] Davis & Geck, Danbury, CT.

CPB = cardiopulmonary bypass; PGA = polyglycolic acid.

dium) to a biodegradable material (polyglycolic acid [PGA]; Davis & Geck, Danbury, CT) when each was used as a pericardial substitute with and without CPB. Through this comparison, we hoped to improve our understanding of the adhesion process and the influence of CPB on formation of adhesions to the native pericardium and implanted pericardial substitutes. The third objective of this study was to observe the frequency and severity of calcification [24, 25] when a biological pericardial substitute was used.

Material and Methods

Although most experimental studies of pericardial substitution have been conducted in dogs or small laboratory animals, we chose weanling sheep for our investigations. Weanling sheep are growing animals that exhibit strong healing reactions and that have demonstrated minimal susceptibility to infection. In these experiments, the pericardial substitute was implanted for 2 to 12 months. Forty-five sheep were assigned to the study as detailed in Table 1.

All sheep were anesthetized with sodium thiopental, intubated, mechanically ventilated, and maintained with oxygen and halothane. The heart was approached through sternotomy or right or left thoracotomy. For experiments with CPB, 1.5 mg/kg heparin was administered. Procedures were performed on a beating heart, using the right carotid artery for systemic arterial return and the right atrial appendage (single cannula) for venous drainage. The CPB was maintained for 30 minutes, followed by removal of a segment of the anterior part of the pericardium and subsequent patching of the defect with a 3 × 2-inch (7.62 × 5.08 cm) sheet of the experimental graft. All grafts were stitched with interrupted, nonabsorbable sutures to facilitate future delineation of the grafts. Thoracotomy was closed in the usual fashion, and air was removed with a chest tube. All animals received postoperative antibiotics for three days. At various intervals after implantation, animals were killed and the implant tissue sites were examined grossly and microscopically for the presence of adhesions and the nature of the tissue response to implantation.

Results

Studies Without Cardiopulmonary Bypass

The microscopic and macroscopic results obtained with glutaraldehyde-fixed bovine pericardium from mixed sources are shown in Table 2 and Figure 1. Most bovine pericardial substitutes showed no adhesions to the heart or lung when CPB was not used (Table 2). Only 1 animal had focal calcifications, which could have resulted from healed infection. One animal had a rolled and thickened pericardial substitute, which may also have been the result of infection. Remarkably, nearly all animals showed no adhesions on macroscopic observation. The PX was smooth and shiny and separated from the heart and lung without difficulty (Fig 1). Although the PX can be differentiated from the native pericardium, it is just as smooth.

Table 2. Macroscopic and Microscopic Results Obtained in Sheep Implanted With Glutaraldehyde-Fixed Bovine Pericardium Without Cardiopulmonary Bypass

Implant Duration (mo)	Pericardial Substitute	
	Macroscopic Observations	Microscopic Observations
2	Thickened but pliable; adhesions	Fibromembranous adhesions; focal granulomatous inflammation
3	Smooth and thin; loose adhesions to the heart	Covered by thin fibrous sheath and fibromembranous adhesions
3	Rolled and thickened with fibrinous exudate mildly adherent to the heart	Collagen breakdown; chronic inflammatory reaction
3	Normal; no adhesions	Normal collagen structure
3	Focal calcification; no adhesions	Cellular reaction
5	Normal; no adhesions	Normal collagen structure
6	Normal; no adhesions	Normal collagen structure
7	Normal except for very mild adhesions	Intact, fibromembranous adhesions
7	Normal; no adhesions	Minimal cellular reaction

Table 3. Macroscopic and Microscopic Results Obtained in Sheep Implanted With Different Combinations of Dexon (PGA) and Maxon Without Cardiopulmonary Bypass

Implant Patch Type	Duration	Pericardial Substitute	
		Macroscopic Observations	Microscopic Observations
PGA-Maxon	134 days	Mild adhesions; separated easily except at base of heart	All FB replaced by autologous collagen
Maxon-PGA	135 days	Pliable adhesions only at base of heart; easily separated	No evidence of FB (absorbed)
Thick mesh	77 days	Mild adhesions, mostly at base of heart	Autolysis; one layer of collagen
PGA-Maxon	151 days	Very thin, soft and pliable (almost transparent); no adhesions	Slight FB; Maxon still evident
Maxon-PGA-Maxon	121 days	Soft and pliable; mild adhesions separated easily, native pericardium adherent	Triple layers; grain cells present
PGA-Maxon-PGA	114 days	No adhesions to heart; adhesions only to lung	PGA disappeared, only Maxon observed in the middle
PGA-Maxon	73 days	No adhesions to heart; very thin material	Two layers; giant cells; Maxon more prominent
Maxon-PGA	198 days	Very thin, soft, pliable; adhesions only to lung	Thin layer of collagen; no FB
Maxon-PGA-Maxon	53 days	No adhesions; smooth, pliable, and looks like native pericardium	Three layers seen
PGA-Maxon-PGA	62 days	No adhesions; soft, pliable	Three layers observed; FB present in all three layers
PGA-Maxon	4 mo	Mild adhesions; soft, pliable, almost transparent	No FB; smooth surface; autologous collagen
Thick mesh	3.5 mo	Very thin, almost transparent, mild to moderate adhesions	No FB; smooth surface with autologous collagen (very thin)
Maxon-PGA-Maxon	4.5 mo	Mild adhesions to base of heart, very thin layer of collagen	Most of FB replaced by autologous collagen
Maxon-PGA	7 mo	Mild adhesions, very thin, low strength	Thin layer of collagen; no FB
PGA-Maxon	7 mo	Mild to moderate adhesions, very thin, low strength	Thin layer of collagen; no FB
PGA-Maxon-PGA	5 mo	Soft, but severe epicardial reaction and adhesions; difficult to separate	Three thin layers of collagen seen; FB still evident
Maxon-PGA-Maxon	4.5 mo	No adhesions, soft, pliable; xenograft thick and calcified	Thin collagen layers; FB still evident
Maxon-PGA	5 mo	No adhesions; soft, pliable, very thin	Thin collagen layers; FB still evident

FB = foreign body; PGA = polyglycolic acid.

The epicardium (Fig 1A) is also as smooth as the left atrial appendage.

Different combinations of Dexon (PGA) and Maxon absorbable mesh materials implanted without CPB also showed no adhesions or very mild adhesions in most animals (Table 3, Fig 2). Mild adhesions could be sepa-

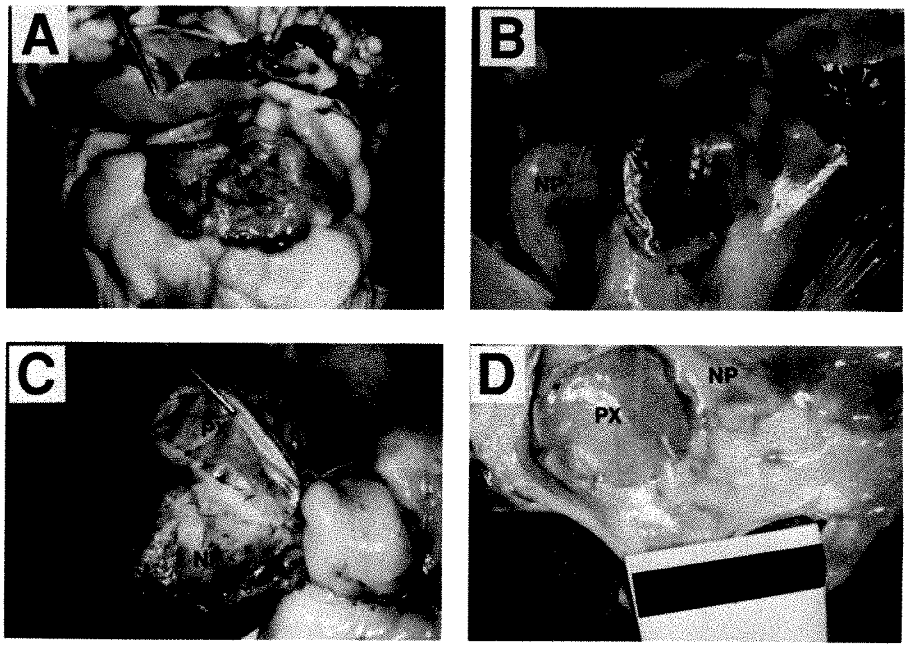

Fig 1. Xenograft pericardium used as a pericardial substitute without cardiopulmonary bypass. (A) The pericardial xenograft is as smooth as the epicardium and the left atrial appendage (LAA). *(B–D) The pericardial xenograft* (PX) *is smooth and shiny and can still be differentiated from the native pericardium* (NP). *It is easily separated from the heart.*

rated by blunt dissection. The absorbable mesh was replaced with autogenous collagen, which was very pliable but had little strength. At less than 3 months, some of the Maxon material remained, with the foreign material surrounded by severe foreign body reactions with giant cells. In animals with implants for more than 5 months, foreign body reaction was virtually absent (Fig 2). In Figure 2A, the pericardium is smooth and shiny, and the now very thin PGA patch is surrounded by smooth native pericardium. Figure 2B shows the native pericardium with epicardial layer and native collagen fibrin. Figure 2C shows PGA layers under higher magnification. The epicardial layer is very smooth, and newly formed collagen fibrin has replaced most of the PGA material. A group of foreign materials is surrounded by giant cells. Figure 2D shows PGA layers under lower magnification. The smooth epicardial layer is formed of new collagen fibrils. More foreign bodies and strong foreign body reactions with multiple giant cells are apparent toward the outer areas of the patch. As shown in Table 3, consistent results were obtained whether Maxon, Dexon (PGA), or a combination of the two absorbable materials was used. Although Dexon (PGA) and Maxon combinations were accompanied by severe foreign body reactions, they did not adhere to the heart or the lung and were replaced with autogenous collagen, which became very thin with time.

Fig 2. Combinations of Dexon (polyglycolic acid) and Maxon absorbable materials explanted after 3 months. (A) Pericardium is smooth and shiny, and the now very thin PGA patch (a) *is surrounded by smooth native pericardium* (Np). *(B) Native pericardium with epicardial layer* (EPL) *and native collagen fibrin* (CF). *(×100.) (C) Polyglycolic acid layers under greater magnification (×200). The EPL is very smooth, and newly formed collagen* (CF) *has replaced most of the polyglycolic acid material. A group of foreign body* (FB) *materials is surrounded by giant cells* (GC). *(D) Polyglycolic acid layers under lower magnification (×150). The smooth EPL is formed of new collagen fibrils. More FBs and strong FB reactions with multiple* GC *are seen toward the outer areas of the patch. (B–D were stained with hematoxylin and eosin; all magnifications are before 29% reduction.)*

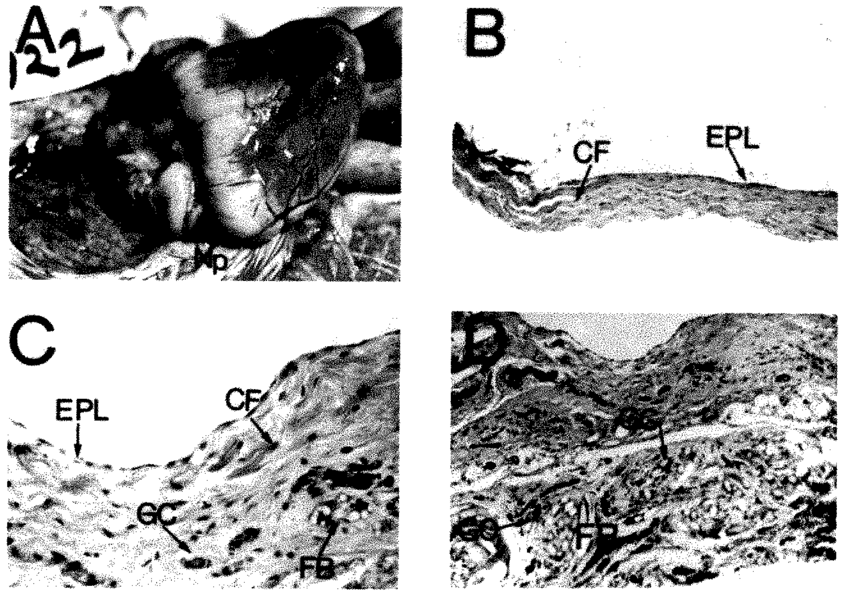

Table 4. Macroscopic and Microscopic Results Obtained in Sheep Implanted With Xenograft (Periguard) With Cardiopulmonary Bypass

Duration (mo)	Pericardial Substitute	
	Macroscopic Observations	Microscopic Observations
2	Soft, pliable, slightly thickened	Small amount of fibrin on membranous side
2	Thin with loose, membranous adhesions	Chronic inflammatory reaction
2.5	Severely adherent to the sternum and heart; soft, pliant, gross epicardial reaction	Thick fibrinous layers on both sides of Periguard
3	Severely adherent to sternum and heart, soft and pliant; thick fibrin layer over heart; identification of coronary arteries difficult	Periguard surrounded by cellular reaction, newly formed thick collagen fibers; FB reaction around suture material
3	Severe adhesions to sternum and heart; separation not possible	Thick collagenous tissue on both sides of implant
3	Severe adhesions to sternum and heart; thick fibrous layer over graft	Severe FB reaction
3	Shrunken, severely calcified, difficult to see coronary arteries	Severe FB reaction; thick fibrin on both sides of implant
6	Thickened but pliable	FB reaction; new collagen deposition on surface
9	Eaten away; no calcification	Severe histocytic and giant cell reaction
9	Slight calcification; shrunken and adherent to the heart	Cellular and degenerative reaction
12	Soft, pliable, nonadherent	Almost normal
12	Shrunken and slightly adherent to the heart	FB reaction

FB = foreign body.

Studies With Cardiopulmonary Bypass

In groups 3 and 4, we compared bovine Periguard (Bio Vascular, St Paul, MN) with PGA when CPB was used. The results are shown in Table 4 for bovine Periguard (group 3) and in Table 5 for PGA both with and without Dacron (group 4). The first six experiments in group 3 combined pericardial substitution with additional procedures that necessitated use of CPB (mainly work on mitral valves). When the animals were killed and inspected, the epicardium was never smooth and a layer of fibrin was always present (Table 4 and Fig 3). In some cases, the layer was very thick (Fig 3A), and it was difficult to separate the pericardium from the heart. Even with sharp dissection, identification of the coronary arteries was difficult (external side view, Figs 3B, 3C). The strongest adhesions occurred at the base of the heart and above the left atrial appendage (Fig 3D). These observations led us to believe that the same pericardial substitute would behave differently when CPB was used.

The next 6 animals in group 3 received pericardial substitutes during a 30-minute CPB period during which no other experimental procedures were performed. These animals were later used for direct comparison with the animals in group 1 (PX without CPB) and as controls for group 4 animals, which received a different substitute material.

Comparison with group 1 animals showed that use of CPB indeed made a great difference (see Table 4). Practically all of the Periguard was adherent to the heart, with a fibrinous layer of varying thickness lying over the heart (Fig 4). Figure 4A shows the PX separated from the heart with sharp dissection. A gelatinous layer covers the heart. Figure 4B is a microscopic view showing the PX. A strong foreign body reaction is visible, with giant cells and a thick fibrin layer. Figure 4C shows the very severe adhesions. Again, the material could be separated with sharp dissection, but not without causing trauma to the heart. Still no myocardium could be seen. Figure 4D shows the very thick fibrin layer with new vessels over the myocardium.

In some animals, the Periguard was calcified. Microscopically, unless and until degeneration of the substitute material occurred, this pericardium was acellular. The host cells could not penetrate this pericardial substitute. Figures 5A and 5C show examples of the severe adhesions formed between the pericardium and the epicardium when CPB was used. Although with sharp dissection the PX could be separated from the heart, the epicardium was covered with a thick gelatinofibrous layer that masked the coronary arteries. The PX was practically acellular (Fig 5B); no cell migration occurred through the material. Strong foreign body reaction existed on both sides of the PX, with no cellular migration through the substitute material. The most severe foreign body reaction was seen at the external aspect of the PX. Higher magnification (Fig 5D) shows the practically acellular PX being "chewed" by giant cell activity. There was a strong cellular reaction on both sides but not within the PX, and the intensity of the foreign body reaction varied widely. Although it could be separated from the epicardium by sharp dissection through the loose contact line of separation, there was a thick fibrin layer severely adherent to the heart itself.

In nearly every case, the native pericardium was severely or mildly adherent, and sharp dissection was needed to achieve separation. The base of the heart was more plagued by adhesion than the apex for both native pericardium and pericardial substitute.

In group 4 animals implanted with PGA with CPB (the first four experiments shown in Table 5), the PGA is slightly thickened. With blunt dissection the heart could be separated from the sternum with far less effort than that required to separate the PX of groups 1 and 2 (Fig 6). The anatomy of the heart was readily apparent (Fig 6A). The single layer of PGA used in this group was totally absorbed within 3 months for almost all animals and was replaced with a very thin layer of host collagen. This layer prevented adhesions between the lungs and the heart. Figure 6B shows the PGA layer, which was replaced with

Table 5. Macroscopic and Microscopic Observations for Sheep Implanted With Polyglycolic Acid and Polyglycolic Acid + Dacron Mesh With Cardiopulmonary Bypass

Material	Duration (mo)	Pericardial Substitute	
		Macroscopic Observations	Microscopic Observations
PGA	3	Thin, pliable, easily separated from sternum; mild adhesions to the heart; native pericardium more adherent than usual	Few islands of PGA surrounded by layers of collagen similar to the native pericardium; mild FB reaction
PGA	3	Soft, pliable, moderate adhesions; mild, jellylike fibrin layer covering the epicardium	Areas of fine collagen fibers surrounding a few remnants of PGA; mild FB reaction
PGA	4	Mild adhesions; separated easily with finger dissection	No remnant of PGA; collagen fibers similar to native pericardium; mild FB reaction
PGA	4	Soft, pliable, thin membrane easily separated with blunt dissection	Few PGA remnants; fine collagen tissue replacing the PGA; mild FB reaction
PGA + Dacron	3	Transparent; lung separated easily from the heart; partially absorbed; Dacron less adherent to sternum and more adherent to heart	No remnant of PGA; very thin layer of collagen arranged in a laminar fashion similar to the native pericardium; severe FB reaction around Dacron fibers
PGA + Dacron	2.5	Lung could be separated easily from the heart; thin, transparent layer easily separated, giving good access to heart and coronary arteries; Dacron forming a thick and strong layer separated with finger dissection	Remnant of PGA with mild cellular reaction; laminated collagenous tissue and few giant cells, thin fibrous tissue has replaced PGA; severe FB reaction surrounding Dacron filaments

FB = foreign body; PGA = polyglycolic acid.

native collagen. When this thin, transparent gelatinous layer was removed (Fig 6C), the coronary arteries were clearly visible and readily accessible (Fig 6D). As shown in Figure 6C, even the native pericardium adhered to the heart when CPB was used, but it too could be separated with sharp dissection relatively easily. The lung could be peeled away very easily from the heart with the finger. We therefore conclude that the PGA material succeeded in preventing adhesions.

The last two animals in group 4 (Table 5) received a combination of nonabsorbable mesh (Dacron) and PGA. The first demonstrated severe adhesion of the Dacron to the heart; in the second, the Dacron mesh became embedded in the host collagen and was easily separated from the heart. Figure 7A shows the sternum of an animal opened 3 months after operation. The lung is apparent on the left side toward the head of the animal. The PGA and Dacron mesh cover the heart on the lower part. The adhesions between the structure and the sternum were very loose and could be separated easily with finger dissection. The lung was peeled off the heart very easily, clearly exposing the anatomy of the lung (Fig 7B). This easy and clean separation could never be accomplished before without use of PGA mesh. The Dacron mesh and the PGA could be separated with blunt dissection (Fig 7C), and the Dacron gave strength to the PGA. As shown in Figure 7D, the Dacron can create a very strong foreign body reaction with multiple giant cells. Dacron fibers are also visible. Without the PGA, the Dacron mesh adheres very strongly to the heart, making it impossible to separate the two without causing severe damage to the epicardium.

The Dacron in this case provided the strength needed by the composite pericardial substitute. Microscopic ob-

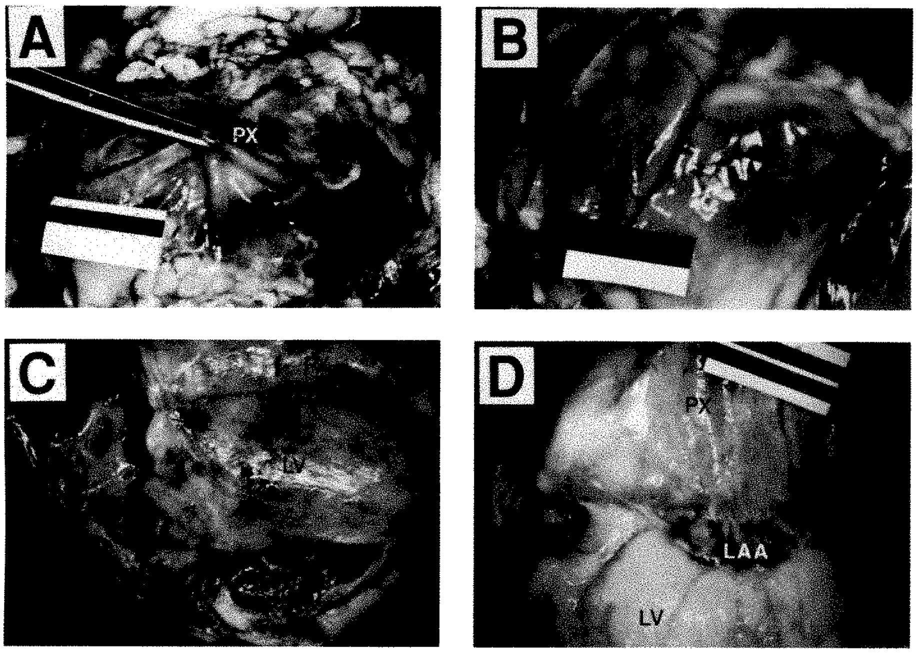

Fig 3. Pericardial xenograft (PX) implanted with cardiopulmonary bypass explanted after more than 3 months. (A) The material (PX) has become very thick, and separation from the heart is difficult. (B, C) Even with sharp dissection, identification of the coronary arteries is difficult (external side view, LV = left ventricle). (D) The strongest adhesions are at the base of the heart and above the left atrial appendage (LAA).

servation revealed a strong foreign body reaction to the Dacron filaments (Fig 7D) and almost complete PGA absorption with replacement by host collagen.

Comment

This basic research into pericardial substitution suggests the importance of understanding the causes and mechanisms of adhesions before considering materials for implantation. Initially it appears preposterous to use biodegradable materials to cover the heart, because the materials may create a very strong foreign body reaction. We therefore approached this study with great caution. We expected that the reaction to absorbable mesh (which permits free migration of cells and nutrients through its large pores) would help us to understand the contribution of such exchange to adhesion formation. We were very surprised to see that the different combinations of Maxon and Dexon (PGA) mesh in group 2 animals did not adhere to the heart and were replaced by a thin layer of collagen. Furthermore, microscopic examinations revealed that although a strong foreign body reaction was present, the materials did not induce pure inflammation. Once the foreign body was digested by macrophages and multiple giant cells, the reaction subsided, leaving a thin layer of collagen that could actually prevent adhesion formation. Why this combination of materials resulted in such strong foreign body reactions but did not adhere to the epicardium is not well understood at this time.

The relatively excellent results obtained with PX as a

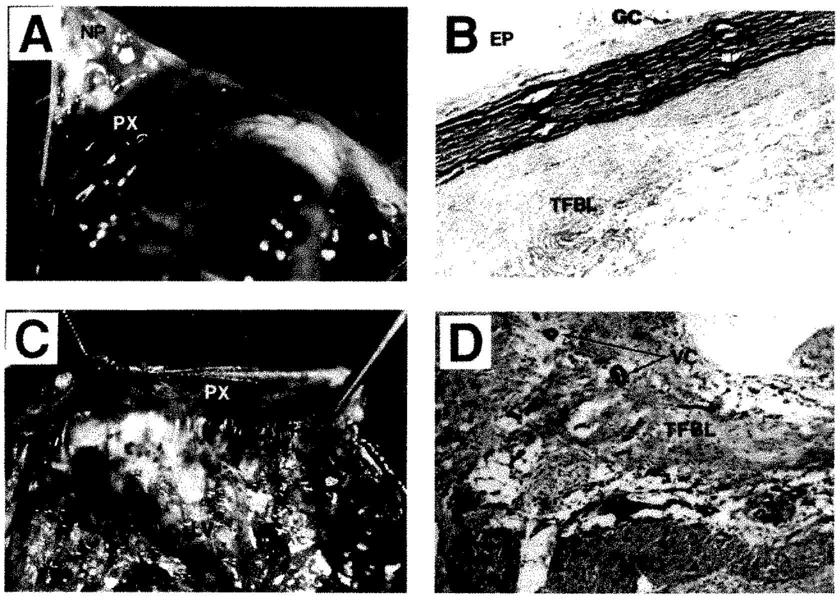

Fig 4. Pericardial xenograft (PX) with cardiopulmonary bypass explanted at 3 months. (A) Pericardial xenograft separated from the heart with sharp dissection (NP = native pericardium). A gelatinous layer covers the heart. (B) Fibrinous layer on the epicardial side (EP) with giant cells (GC) and thick fibrin layer (TFBL). The PX is seen as an acellular layer, with thick fibrinous reaction on both sides (×100). (C) Very severe adhesions. The material could be separated with sharp dissection, but not without causing trauma to the heart. Still no myocardium is seen. (LV = left ventricle.) (D) Microscopic view (×150) showing the TFBL with new vessels (VC) over the myocardium (MYC). (B and D are stained with hematoxylin and eosin; all magnifications are before 29% reduction.)

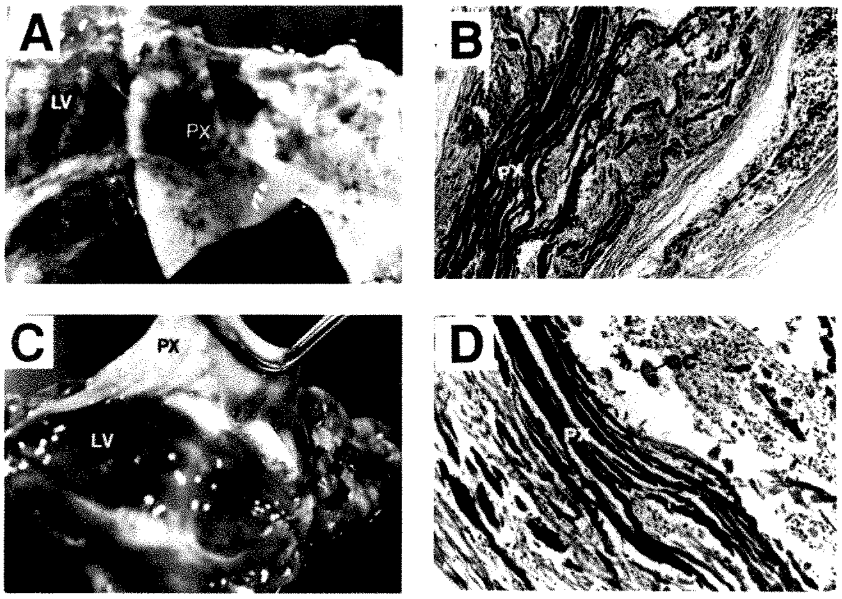

Fig 5. Periguard pericardial xenograft (PX) with cardiopulmonary bypass. (A, C) Severe adhesions formed between the pericardium and the epicardium when cardiopulmonary bypass was used. Although with sharp dissection the PX can be separated from the heart (LV), the epicardium is covered with a thick gelatinofibrous layer that masks the coronary arteries. (B, D) The PX is practically acellular, with no apparent cell migration through the material until it is "chewed" by macrophages and giant cells. There is severe foreign body reaction from the external aspect of the PX. (B, ×150; D, ×200 before 29% reduction; both stained with hematoxylin and eosin.)

pericardial substitute when no open heart CPB procedure is performed are also notable. Blood clotting after CPB might be the reason for such clear differences between results seen with CPB (group 3) and without CPB (group 1). More severe adhesions between the remaining native pericardium and the heart also accompanied CPB. Some investigators [22, 23] have suggested that the pericardium possesses fibrinolytic activity that can prevent blood clotting in the pericardial sac. This activity is a major factor preventing activation of fibrinogen into fibrin—a source of future adhesions.

Preliminary studies in our laboratory suggest that this fibrinolytic activity is also present in the epicardium. The results obtained with the PX in group 3 (with CPB) clearly showed that the use of CPB incurs not only adhesion between the PX and the heart, but also a thick fibrin layer adherent to the epicardium (Fig 3). This fibrin layer makes reoperation very difficult, in agreement with the experience in humans described by Gallo and co-workers [13]. Pericardial xenograft substitutes have generally given excellent results in laboratory animals, but in humans the results have not been entirely satisfactory. Because we reproduced the severe adhesions observed in humans by putting the animals on CPB, we can claim with certainty that the differences previously observed between animals and humans are not species differences but result from the actual operating conditions and damage to the heart and the pericardium incurred at the time of operation.

In addition, we noted that the PX induces a strong foreign body reaction that is characteristically acellular

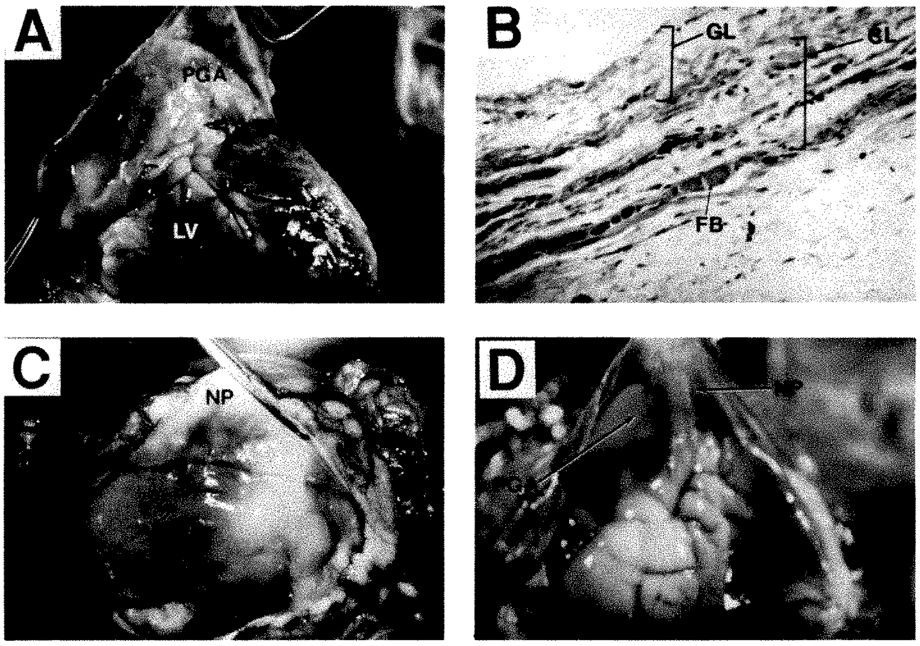

Fig 6. Polyglycolic acid (PGA) as a pericardial substitute with cardiopulmonary bypass. (A) Explanted after 3 months, the PGA is slightly thickened but could be separated with blunt dissection. The anatomy of the heart is readily apparent. (B) Microscopic view (×200 before 29% reduction; stained with hematoxylin and eosin) of the PGA layer, which was replaced with native collagen (CL). A fine gelatinous layer (GL) is present over the PGA removed from the heart. This layer may make separation easier. (C) Loose adhesions are present between the native pericardium (NP) and the PGA, which appears almost transparent. (D) With gelatinous layer removed, anatomy of the heart is clearly seen. (FB = foreign body.)

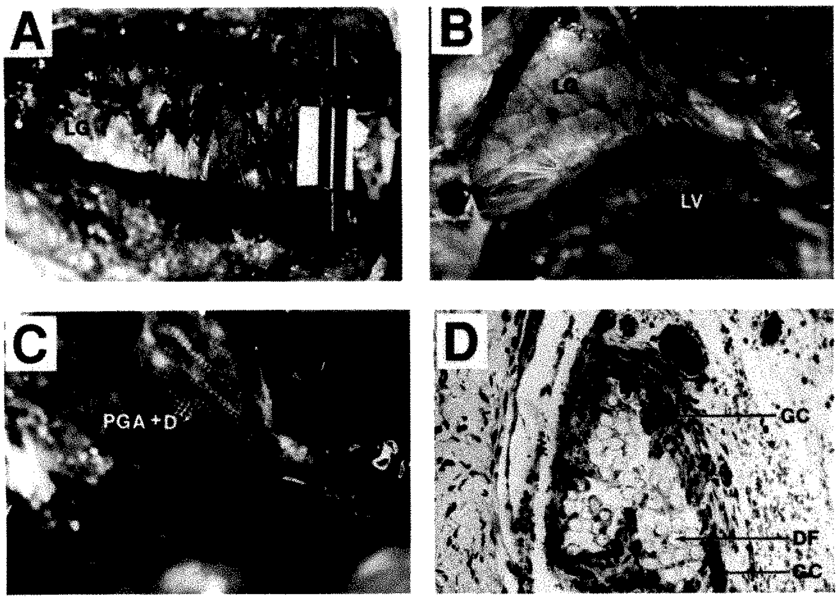

Fig 7. Polyglycolic acid (PGA) and Dacron mesh (large pore) combination with cardiopulmonary bypass explanted after 3 months. (A) Sternum of an animal opened 3 months after operation. On left toward the head of the animal is the lung (LG). The PGA and Dacron mesh (PGA + D) cover the heart on the lower part. Adhesions between the structures and the sternum were very loose and could be separated easily with finger dissection. (B) The lung was peeled off from the heart (LV) very easily, clearly exposing its anatomy. (C) The Dacron mesh and PGA material could be separated with blunt dissection, with the Dacron adding strength to the PGA substitute. (D) Dacron can create a very strong foreign body reaction with multiple giant cells (GC). Dacron fibers (DF) are also visible. Without the PGA, the Dacron mesh adheres very strongly to the heart, making it impossible to separate it without causing severe damage to the epicardium. (Hematoxylin and eosin, ×200 before 29% reduction.)

(Fig 6). Although the macrophages slowly destroy the collagen fibers, they are not free to move from one side of the graft to the other, as they were in groups 2 and 4 (biodegradable mesh). The newly formed collagen is obviously rich in nuclei (Fig 7), proving that it is newly formed collagen. The remaining foreign body is also surrounded by cellular elements because the foreign body exists in multiple small islands. Because the PX is dead tissue, it not only calcifies but also is incapable of participating in local metabolic processes such as replacement of biodegradable materials with newly formed collagen. This study shows that a PX fixed with glutaraldehyde is not an ideal solution to the problem of pericardial substitution.

Through this investigation, we now believe that PGA mesh has a definite advantage as a material to be used for prevention of adhesions between the heart and surrounding tissue. Polyglycolic acid mesh is not yet an ideal pericardial substitute, however, because it has insufficient strength after PGA absorption and cannot adequately protect the heart from injury at the time of reoperation. In light of this, we implanted PGA mesh combined with a very-large-pore Dacron mesh into the last 2 animals in group 4 (animals 5 and 6, Table 5). The Dacron was intended to contribute strength after the PGA was absorbed while still permitting free migration of cellular elements through the pores. Although 3 months later we saw adhesions in the first animal, in the second animal the newly formed "pericardium" could easily be removed from the heart with finger dissection. The anatomy of the heart was readily visible, no thick layer of fibrin covered the epicardium, and the new pericardial layer demonstrated substantial strength

Conclusions

Based on results obtained in weanling sheep using pericardial substitutes without and with CPB, we conclude the following:

1. After 3 to 9 months, the xenograft pericardium (Periguard) implanted without CPB showed no adhesions, but PX implanted with CPB showed severe adhesions to the heart, making reoperation nearly impossible. Pericardium xenograft is therefore not practical as a pericardial substitute if CPB is used. The discrepancies between clinical experience with xenograft pericardium and the results of animal experiments performed without CPB are due to the effects of CPB on adhesion formation. Future experimental evaluations of potential pericardial substitutes must use CPB.
2. Pericardial substitute made of biodegradable PGA mesh was replaced in 3 to 4 months by a thin layer of autogenous collagen that did not adhere to the heart. This very thin collagen layer allowed the lung and other surrounding tissue to be peeled away from the heart. The anatomy of the heart was still visible, and the coronary arteries were not covered with a thick layer of fibrin. Therefore, PGA may be an excellent material for prevention of adhesions.
3. A pericardial substitute must not inhibit the normal exchange of nutrients and cellular elements between the heart and the surrounding tissue. In this regard, PGA mesh may be an excellent material for preventing adhesions between the lung and chest wall or between the heart and the surrounding tissue. However, PGA mesh needs the additional strength of a nonabsorbable material before it can be considered an ideal pericardial substitute.

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Reoperation After Pericardial Closure With Bovine Pericardium

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To reduce the risk of damage to the anterior surface of the right ventricle after resternotomy, it has been recommended that the pericardium be closed with a patch after open heart operations. We have examined 4 patients undergoing resternotomy for the third time 3 to 8 years after bovine pericardium valve replacement. On each occasion the pericardium was closed with a patch of bovine pericardium. In all cases, the patch was frozen to the inner aspect of the sternum, increasing the difficulty of resternotomy. Histological examinations of the patches confirmed dense fibrous connective tissue, patchy calcification, and foreign body giant cell reaction. Bovine pericardium appears to increase the difficulty of repeat cardiac operations. We recommend its use be discontinued.

(Ann Thorac Surg 1989;48:813–5)

Repeat cardiac operations have become increasingly common in recent years not only in valvular diseases [1, 2] but also in coronary artery operations [3]. Technical difficulties that may be encountered lead not only to increased morbidity [1] but also to mortality [4]. To minimize pericardial adhesions and thus ease reentry at a subsequent procedure, primary pericardial closure has been advocated [5]. However, this may not be possible in all cases due to pericardial contraction and the fear of cardiac tamponade [6]. In such cases, pericardial substitutes have been applied to shield the anterior surface of the heart from direct adhesion to the sternum. These include silicone rubber, silicone polyester, polyfluoroethylene, and heterogeneous pericardium [7]. We report our recent experience of resternotomy 3 to 8 years after the implantation of glutaraldehyde-preserved bovine pericardium as a pericardial substitute.

Patients and Methods

Four patients who underwent valve replacement and closure of the pericardial cavity with glutaraldehyde-preserved bovine pericardium required reoperations for valve failure 3 to 8 years later. Over the same period, 113 bovine pericardial patches were used to reconstruct the pericardium after second repeat valve replacement operations. Table 1 summarizes patients' details and the types of operations performed.

Resternotomy was performed using an oscillating saw. Dense adhesions were found not only to the posterior surface of the sternum but also to the epicardium. Patient and cautious sharp dissection enabled successful freeing of the heart in all patients. Intracardiac procedures were performed uneventfully. Specimens of the previously implanted bovine pericardium were removed for histological examination.

Results

There was no operative mortality. All patients made uneventful postoperative recovery.

Histological examination of the bovine pericardium patches removed showed that although the bovine pericardium itself was well preserved structurally, the surrounding tissues demonstrated dense, poorly vascular fibrous connective tissue (Fig 1), foreign body giant cell reaction (Fig 2), and chronic inflammatory cell infiltrate (Fig 3). The tissue reaction was more intense in the patch removed 8 years after implantation compared with that removed 3 years after usage.

Comment

Pericardial adhesions result from serosal injury and spilled blood during cardiac operations [8]. Closure of the

Table 1. Pericardial Substitute

Patient No.	Age (yr)	Sex	Operation	Previous Operations	Length of Implantation (yr)
1	34	F	Redo MVR	CMV, MVR × 2	3
2	46	M	Redo AVR, MVR	AVR, MVR	4
3	46	M	Redo AVR, MVR	Repair coarctation, AVR, MVR	5
4	53	M	Redo AVR	AVR × 2	8

AVR = aortic valve replacement; CMV = closed mitral valvotomy; MVR = mitral valve replacement.

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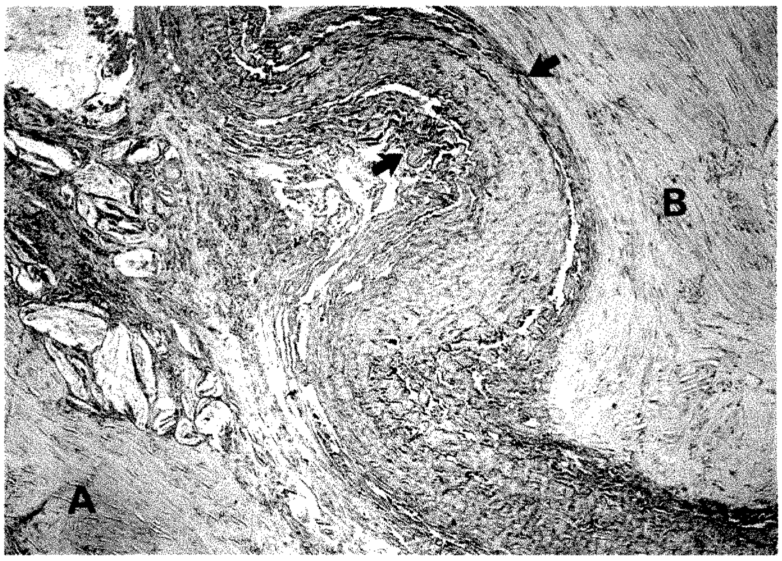

Fig 1. Bovine pericardium (arrows) *with dense substernal* (A) *and epicardial* (B) *scars. (Hematoxylin and eosin, ×100 before 43% reduction.)*

pericardium, if feasible, reduces such adhesions and aids reoperations [5, 6]. This has not been found to affect graft patency after coronary artery bypass grafting [9], postoperative blood loss, or the requirement for reexplorations [10]. Conversely, pericardial closure prevented cardiac tamponade as most postoperative bleeding appeared to originate from extrapericardial sources [6].

The pericardium may shrink substantially within one to two hours of exposure during cardiac operations [6]. This may make it impossible to effect primary pericardial closure without compressing the heart, with harmful hemodynamic consequences. Many pericardial substitutes have been advocated to allow closure without tension and to ease resternotomy by preventing direct adhesion of the heart to the posterior surface of the sternum. These include bovine and equine glutaraldehyde-preserved xenografts [11, 12], expanded polytetrafluoroethylene [13], siliconized Dacron [14], Silastic [15], and autologous fascia lata [16]. Of these, polytetrafluoroethylene appears to be a satisfactory substitute [7], especially in children requiring multiple corrective procedures [13].

Since the initial reports of favorable results in experimental animals [17], bovine pericardium has become increasingly used in achieving pericardial closure. It soon emerged both in experimental animals [11] and in clinical application that pericardial adhesions still occurred from intense inflammatory reaction [18, 19]. Calcification can result [11]. This may be the result of mechanical distortion [20] or the use of preservatives [19]. Our experience with glutaraldehyde-preserved bovine pericardium as a pericardial substitute as presented here confirms these observations. These adhesions not only complicate resternotomy but also make identification of coronary anatomy difficult [12]. Use of autogenous tissues, including fascia lata [16], may reduce the intensity of such inflammatory response. In our institution, bovine pericardium is no longer used for such a purpose. A controlled study is thus not feasible. No other pericardial substitutes have been

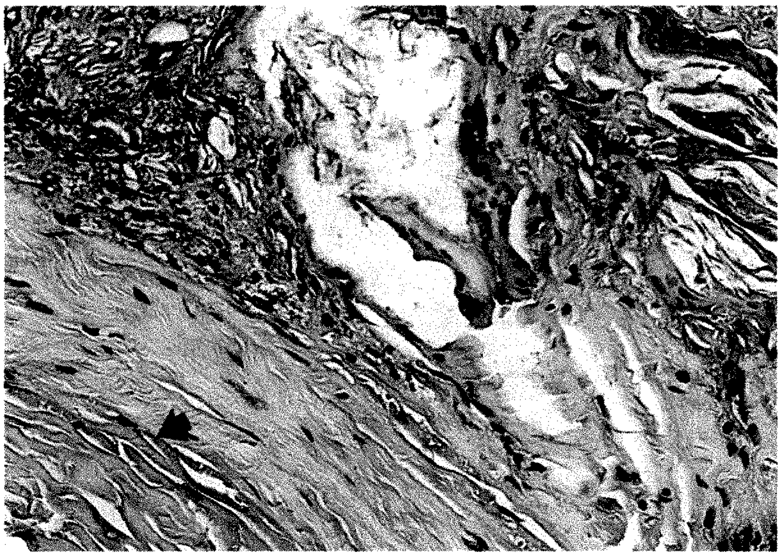

Fig 2. Substernal foreign body giant cell reaction adjacent to bovine pericardial patch (arrow). *(Hematoxylin and eosin, ×265 before 43% reduction.)*

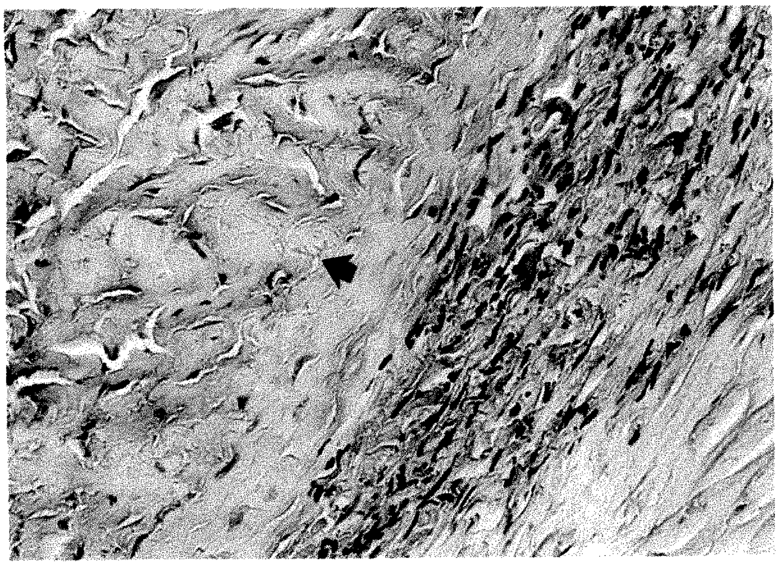

Fig 3. Chronic inflammatory cell infiltrate in scar adjacent to pericardial patch (arrow). *(Hematoxylin and eosin, ×265 before 43% reduction.)*

used in our unit. However, our experiences of reoperating on patients who previously had bovine pericardial substitute place us in a favorable position to describe the difficulties encountered, as we appear to have a higher frequency of repeat valve operations than other groups [1].

Although technically more demanding, resternotomy after a previous cardiac operation with the pericardium left open may not be associated with a higher operative mortality provided a careful technique is adopted [2]. When reoperation appears likely at the first procedure, primary pericardial closure should be attempted. This may be aided by methods of traction to prevent pericardial contraction after its incision [6]. The use of pericardial substitutes in cardiac surgery remains doubtful, and caution is advised in their wider application [7].

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Operation for Acute Postinfarction Mitral Insufficiency Using Continuous Oxygenated Blood Cardioplegia

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Patients with acute-onset mitral insufficiency and cardiogenic shock after myocardial infarction have a high incidence of operative death and morbidity. Patients with ventricular dysfunction, myocardial ischemia, and limited cardiac reserve undergoing an urgent operation represent a challenge to modern methods of myocardial protection. To improve results of operation a new technique was devised with continuous infusion of cold oxygenated blood cardioplegia during the entire cross-clamp period. Between 1984 and 1988, 19 consecutive patients with severe mitral regurgitation and cardiogenic shock (systolic blood pressure less than 60 mm Hg) after myocardial infarction underwent urgent myocardial revascularization, mitral valve replacement, or both. Left ventricular ejection fraction was less than 40% in 16 of 19 patients. All patients had suffered myocardial infarction within 4 weeks of operation and underwent an urgent operation within 24 hours of the onset of hemodynamic compromise. Severe three-vessel coronary artery disease was present in 16 of the 19 patients. A continuous infusion of blood cardioplegia was instituted at aortic cross-clamping and continued throughout the cross-clamp period. Infusion of continuous blood cardioplegia was also instituted through each completed distal vein graft. Myocardial septal and left ventricular apical temperatures were maintained at 10° ± 2°C throughout the cross-clamp period. There were two in-hospital deaths (mortality, 10.5%) and low output syndrome was present in 10 patients (53%). At a mean follow-up of 2.5 years, there was one late death and 14 of the 16 remaining patients were in New York Heart Association functional class I or II. Continuous infusion of cold blood cardioplegia may be technically more demanding than traditional intermittent infusion; however, it may offer improved myocardial protection in high-risk patients undergoing an urgent operation. Additional randomized controlled clinical trials are necessary to allow improvement in mortality and morbidity in this complication of myocardial infarction.

(Ann Thorac Surg 1989;48:816–9)

Intermittent infusion of cold blood cardioplegia allows for oxygenation of myocardial tissues at intervals, but it renders the myocardium anoxic when the infusion is interrupted. This allows for rewarming of myocardial tissue with localized ischemia, inhomogeneous cooling, and anaerobic metabolism. Although the heart is protected by hypothermia and cessation of electromechanical activity, anaerobic myocardial metabolism persists during aortic cross-clamping. Myocardial protection may be adequate for hearts with uncompromised cardiac reserve in the absence of preoperative myocardial ischemia. Urgent myocardial revascularization or mitral valve replacement (or both), impaired preoperative ventricular function, and operation after myocardial infarction are independent and incremental risk factors for operative mortality and morbidity. Patients undergoing an urgent operation for postinfarction mitral regurgitation and cardiogenic shock are at a high risk for operation, with a reported mortality rate of 21% to 85% [1–10].

Continuous infusion of oxygenated cold blood cardioplegia may be of benefit in high-risk patients. Uniform myocardial cooling, washout of metabolites, maintenance of consistently low myocardial temperatures without major fluctuations, and continuous delivery of oxygen and nutrient flow to the microcirculation may optimize myocardial protection by meeting metabolic demands during aortic cross-clamping. A prospective trial of this new technique was instituted in a consecutive series of high-risk patients undergoing urgent myocardial revascularization or mitral valve replacement, or both, for postinfarction mitral regurgitation and cardiogenic shock.

Material and Methods

Patient Population

Between October 1984 and December 1988, 19 consecutive patients (13 male, 6 female; average age, 64 ± 10 years) were seen with acute-onset mitral regurgitation and cardiogenic shock within 4 weeks after myocardial infarction and underwent urgent myocardial revascularization or mitral valve replacement, or both.

Cardiogenic shock was defined as hemodynamic compromise with systolic blood pressure less than 60 mm Hg requiring intraaortic balloon pump counterpulsation or large doses of inotropic support (dopamine, 15 to 25 μg/kg normal body weight, and adrenaline, 1 to 2 mg/kg), or

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both methods, to maintain systemic perfusion pressure greater than 60 mm Hg. Seven patients required intraaortic balloon pump assistance preoperatively, and all patients were treated with dopamine or adrenaline, or both. All patients had Swan-Ganz catheters inserted preoperatively for hemodynamic management and proceeded to operation within 24 hours of onset of hemodynamic compromise. Preoperative cardiac catheterization with left ventricular angiograms and coronary angiography was performed in all patients.

The patients were all functioning at New York Heart Association class IV and had left ventricular ejection fraction graded by angiography. There were 3 patients with left ventricular grade II, 4 with grade III, and 12 with grade IV.

Before operation all patients had two-dimensional echocardiography, and at operation the echocardiographic findings were confirmed. There were 10 patients with anterior papillary muscle rupture, 5 with posterior papillary muscle rupture, and 4 without rupture but with severe papillary muscle dysfunction.

There was one mitral valve repaired in this series; 4 patients underwent mitral valve replacement (MVR) alone. The other 15 patients had MVR plus coronary artery bypass grafting (CABG). There were 6 patients with MVR and triple CABG, 6 with MVR and double CABG, and 3 with MVR and single CABG.

The mean time from myocardial infarction to operation was 17.9 ± 9 days. Myocardial infarction was diagnosed by electrocardiogram and enzyme criteria (new Q wave and greater than 7% rise in the level of myocardial-specific isoenzyme of creatine kinase). Anterior myocardial infarction was present in 10 patients, posterior infarction in 5, and anteroseptal infarction in 4.

Operative Technique

Ascending aortic cannulation was performed after heparinization. Both the superior and inferior venae cavae were cannulated and not snared to allow for venous drainage. Moderate hemodilution (hematocrit, 20% to 25%) and systemic hypothermia (28°C) were maintained during cardiopulmonary bypass. Topical hypothermia was not employed. Thermistor probes were placed in the anterior septum and the left ventricular apex to monitor myocardial temperature. Cold high-potassium blood cardioplegia* was infused into the ascending aorta at a pressure of 90 mm Hg immediately after aortic cross-clamping. Electromechanical arrest and cooling to septal temperatures of 10° ± 2°C required 1,200 ± 300 mL of initial high-potassium cardioplegia. Continuous administration of a cold low-potassium blood cardioplegia† through the aortic root was employed to maintain electromechanical arrest and myocardial temperatures less than 15°C throughout the cross-clamp period. Cardioplegia was infused at a rate of 150 mL/min with an aortic root pressure of 80 mm Hg.

The left atrium was incised and the mitral valve was inspected. Anterior and posterior mitral leaflets were excised. The posterior mitral valve apparatus was left intact when posteromedial rupture of papillary muscles was not present. Distal coronary anastomoses were performed before MVR.

Continuous low-potassium blood cardioplegia was infused into each saphenous vein graft after completion of the distal anastomosis using a manifold system of infusion. The mitral valve replacement was performed with continuous cardioplegia delivery simultaneously through the aortic root and each saphenous vein graft. The mitral valve was replaced with interrupted pledgeted 2-0 Tevdec sutures. No attempt was made at valve reconstruction. All proximal anastomoses were performed during a single aortic cross-clamp period [11, 12]. After systemic rewarming to greater than 36°C and evacuation of air, the aorta was unclamped and patients were weaned from cardiopulmonary bypass.

Results

The mean pump time was 88.3 ± 28 minutes and the mean cross-clamp time was 58.6 ± 20 minutes. The volume of cardioplegia administered was 1,200 mL of high-potassium solution (dilution, 1:4 with blood) and 1,856 ± 812 mL of low-potassium crystalloid cardioplegia. The mean serum potassium at the end of bypass was 5.90 ± 0.99 mEq/L (range, 4.1 to 7.5 mEq/L). Two patients had potassium concentrations greater than 7 mEq/L, which were easily controlled with the administration of Lasix (furosemide).

There were two deaths (mortality, 10.5%). One patient could not be weaned from bypass despite intraaortic balloon pump and inotropic support. The other patient with acute postinfarction mitral insufficiency and end-stage coronary artery disease unsuitable for bypass died six hours after MVR plus attempted aortocoronary bypass in low-output state. Low output syndrome, defined as systolic blood pressure less than 90 mm Hg and/or cardiac index less than 2 L/min requiring inotropic and/or intraaortic balloon pump support, occurred in 10 patients postoperatively.

Complications included a transient stroke in 1 patient two days after operation. One patient in cardiogenic shock preoperatively had the sternum and skin left open for two days; the sternum and skin were subsequently closed and he did well. One patient suffered renal failure requiring hemodialysis and recovered renal function before discharge.

Follow-up

Mean follow-up was 2.8 years. One patient died 3 months after operation due to pneumonia. One patient suffered a stroke 2 years after operation and recovered completely. The other patients are alive and well with the following New York Heart Association class: I, 8 patients; II, 6 patients; and III, 2 patients.

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* High-potassium blood cardioplegia consists of 1,000 mL of 5% dextrose in water, 60 mEq KCl, 18 mEq $MgSO_3$, 12 mEq tromethamine, and 20 mL of CPD solution; osmolality, 374 mosm/L; pH, 8.3; temperature, 15°C. It was delivered by a Buckberg-Shiley system in a dilution of 1:4 crystalloid/blood, resulting in a mixture with hematocrit = 22% and $[K^+]$ = 26 mEq/L.

† Low-potassium blood cardioplegia consists of a solution identical to high-potassium blood cardioplegia but with 27 mEq/L of KCl, resulting in a mixture with $[K^+]$ = 7 mEq/L.

Comment

Despite current methods of myocardial protection, operation for acute postinfarction mitral insufficiency in patients with severe hemodynamic compromise or cardiogenic shock is associated with high in-hospital mortality [13–16]. This may be due to resection of the posterior papillary muscles during MVR, which can result in further deterioration in ventricular function [17–21]. The beneficial effects of preservation of the posterior papillary muscles have long been emphasized [17, 18, 22, 23]. In addition, afterload mismatch that develops after MVR when the incompetent valve is replaced with a competent mitral prosthesis [24] may also be a factor. Others [25–28] have stressed the importance of revascularization, which improved the 5-year survival of such patients. Great improvements in surgical results have been demonstrated with transventricular mitral valve repair combined with revascularization [29].

Despite improvements in surgical techniques, acute pump failure continues to plague surgeons operating on these patients, which may imply a need for better myocardial protective techniques. Continuous oxygenated blood cardioplegia was first used by Bomfim and colleagues [30] in a clinical series of patients undergoing aortic valve replacement. Bomfim and colleagues [30] emphasized its potential use for high-risk patients, confirming our subsequent clinical observation (Salerno TA, Lichtenstein SV, unpublished observations). Khuri and co-workers [31] performed intramyocardial pH studies of similar patients and reported excellent results with its use. Continuous oxygenated blood cardioplegia has been used for all surgical procedures at our institution. This series of patients represents the experience of one surgeon (T.A.S.) in which the technique was modified to allow for MVR.

In patients with acute postinfarction mitral insufficiency with mild aortic regurgitation in whom replacement of the aortic valve was not contemplated, the ascending aorta was opened and each coronary ostium was cannulated to deliver continuous blood cardioplegia during the clamping period (unpublished series of 6 successful patients). As each distal coronary artery anastomosis is performed, perfusion of the myocardium is achieved through either the ostia or the saphenous vein grafts, or both, while the mitral valve is replaced. Perfusion of the myocardium in case of MVR and triple coronary artery bypass can also be achieved by perfusion of the grafts alone without the need to perfuse the aortic root, thereby simplifying the procedure.

We recognize the limitations of this study and the need for larger series of patients with longer follow-up periods to fully delineate the beneficial effects of this technique of myocardial protection. To our knowledge, continuous oxygenated blood cardioplegia has not been previously reported for MVR or operation for acute postinfarction mitral insufficiency.

In conclusion, this work reports on improvement in clinical outcome in a subgroup of high-risk patients with acute postinfarction mitral insufficiency. Continued efforts at myocardial preservation may allow for further decreases in mortality for this condition.

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Modified Open Thoracic Rhizotomy for Treatment of Intractable Chest Wall Pain of Malignant Etiology

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We describe the surgical technique of modified open thoracic rhizotomy for treatment of intractable chest wall pain of malignant etiology. In a series of 14 patients, 9 (64%) had an excellent result, 4 (29%) had a good result, and 1 (7%) had a poor result. Successful palliation was made possible by identification with computed tomographic scan or magnetic resonance imaging of the nerve roots involved. Pain control lasted in most patients until death (median, 22 weeks; range, 6 to 45 weeks). The extrathecal procedure described has certain advantages over intradural transection of nerve roots. Indications for performing this procedure are discussed along with other therapeutic options.

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Malignant disease of the lung or chest wall is frequently accompanied by severe pain. The pain may result from encasement by the tumor of the intercostal nerves or the thoracic nerve roots or from erosion or invasion of the spine or ribs. Toward the end of a patient's course, there may be little to offer in the way of treatment for the malignancy itself (eg, operation, irradiation, or chemotherapy). However, the pain continues to escalate. In most instances, pain may be controlled, at least initially, by analgesic and antiinflammatory drugs. However, in many patients, an increasing need for opioids leads to tachyphylaxis, which leads in turn to increased dosages and drug intolerance. Such patients may be seen by neurosurgeons for pain-alleviating procedures.

We describe our experience with 14 patients who underwent a simplified open thoracic rhizotomy. Excellent and lasting pain control was achieved in 9 patients (64%) and substantial relief in 4 patients (29%).

Material and Methods

Patients

Fourteen patients with malignant tumors of the lung or chest wall causing local pain were treated with a simplified dorsal rhizotomy. In all patients the pain was unilateral; in 7 it was confined to the posterior chest wall and spine, whereas in the other 7 it was also referred to the anterior chest. All patients had progressive malignant disease with Karnofsky performance status ranging between 50% and 70%. Nine patients had exhausted antineoplastic therapy, including operation, irradiation, and chemotherapy. In 5 patients, chemotherapy was continued after the procedure described here. Extensive attempts at pain control with various analgesics, including high-dose narcotics, had been made in all the patients, who were considered candidates for operation only after pain control was judged poor or severe side effects had compromised the effectiveness of the medications.

The decision as to which nerve roots to ablate was made on the basis of careful clinical examination. The results of the examination were then correlated with findings on chest roentgenogram, computed tomography, or magnetic resonance imaging. These tests revealed the precise location of the tumor and showed the extent of soft tissue and bone involvement. From these findings we determined which nerves needed ablation. Diagnostic anesthetic blocks were attempted in a few patients, but these proved not to be particularly informative, mainly because of substance diffusion, which created a hypoesthetic area larger than the area served by the compromised nerve roots.

Operation was considered only in patients who had a limited life expectancy and no other sources of pain and who were acceptable anesthetic risks. Pulmonary contraindications were considered to be an arterial carbon dioxide tension greater than 50 mm Hg or an arterial oxygen tension less than 50 mm Hg, or both, or a forced expired volume at 1 second of less than 1 L.

Surgical Approach

Each thoracic spinal nerve consists of autonomic, preganglionic sympathetic, motor, and sensory nerve fibers that fuse at the level of the neural foramen and emerge as a common nerve trunk. Classically, sensory and motor roots were depicted as being completely separated; the sensory roots enter the cord posteriorly, conducted through the posterior nerve root, whereas the autonomic-sympathetic and motor fibers leave the cord anteriorly through the anterior nerve root.

There is now anatomical and physiological evidence that motor roots contain thinly myelinated fibers that are related to pain conduction, and that the sympathetic

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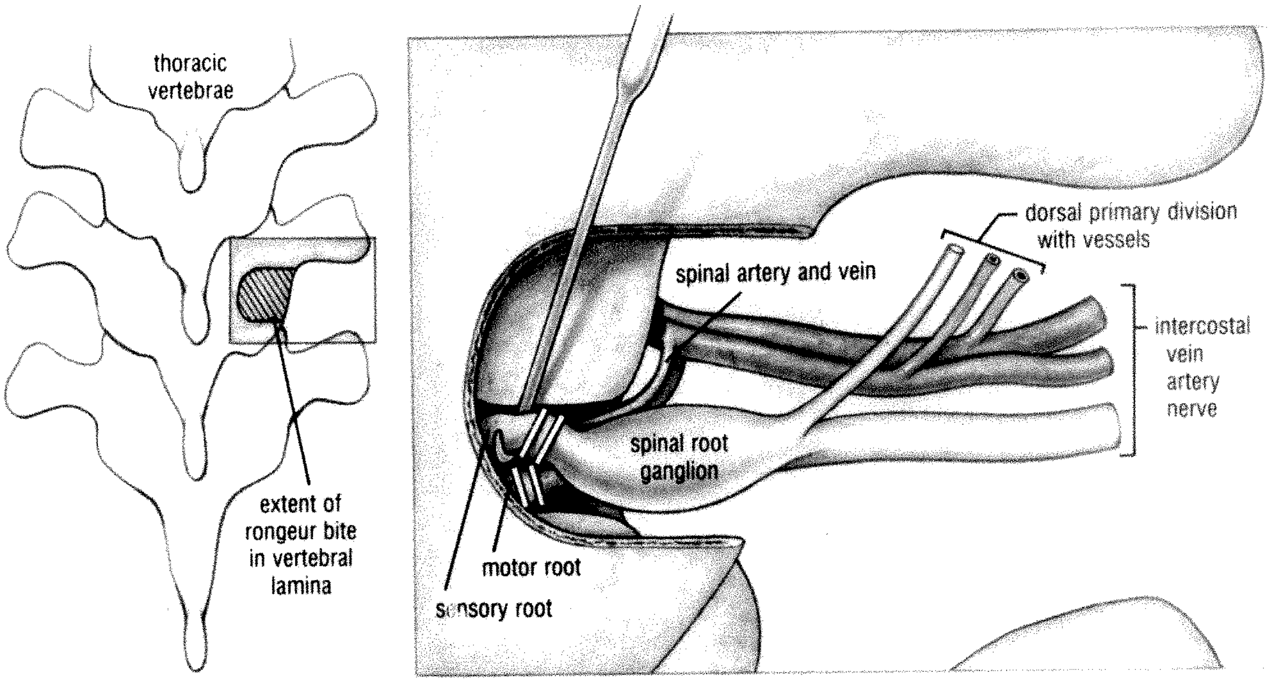

Fig 1. (Left) Site to be exposed to view the neurovascular bundle and (Right) the anatomical structure within the neural foramen. Clips are applied before nerve sectioning to prevent cerebrospinal fluid leakage.

nerves play a role in transmission of visceral pain [1–4]. It is also known that most of the pain-sensitive structures of the spine and paraspinal structures are innervated by branches of the spinal nerves (recurrent meningeal or sinuvertebral nerve) that arise just distal to the dorsal root ganglion. Because of these data, we prefer, when only thoracic segments are involved, to ablate both anterior and preganglionic posterior roots. The roots traverse the spinal subarachnoid space, pierce the dura, and reach the neural foramen. Both anterior and posterior roots are surrounded by dural and arachnoid sleeves. The posterior sensory root is generally thicker than the anterior motor root, and at the level of the intervertebral foramen it swells into a spinal ganglion containing unipolar cells, the origin of sensory fibers. The subarachnoid space stops at the level of the sensory ganglion and the corresponding portion of the motor root, at which point there is a blending of the dura and arachnoid with the connective tissue of the peripheral nerve.

To prevent a cerebrospinal fluid leak from the transected nerve root, we apply a clip proximal to the nerve stump. Preservation of the radicular arteries is important in the watershed zones where the spinal cord blood supply is precarious. The thoracic segments between T-1 and T-4 and between T-11 and L-1 are considered vulnerable in this respect, and arteries running with nerve roots at these segments should be spared. The arteries in the intervertebral foramen run on the anterior surface of the spinal root in the dural investment of the nerve root, and they can be separated and exposed by gentle dissection on the underside of the nerve root.

Our procedure is a modification of an operation described by Scoville in 1965 [5]. In this procedure, only the sensory nerve root was transected, whereas in ours, both anterior and posterior nerve roots are divided for the reasons noted previously.

The procedure is performed using general endotracheal anesthesia. Patients are placed prone on two parallel rolls, leaving the larger part of the chest free to move with respiration. The skin incision is made in the midline, extending one level above and one below the nerve roots to be ablated. Dissection proceeds through the skin and subcutaneous tissue to the deep fascia. The fascia is incised about 2 cm lateral to its attachment to the spinous processes. The erector spinae muscle mass is reflected off the fascia and laterally by stripping the muscles off the spinous processes and laminae. The protuberance at the lamina–pedicle junction and transverse process is exposed (Fig 1).

A lateral roentgenogram is obtained at this juncture to verify the spinal level. A high-speed air drill is used to drill off the outer edge of the lamina just caudal to the transverse process to expose the segmental neurovascular bundle. Usually a 1.5-cm hole is adequate to visualize, clip, and transect the sensory and motor roots within the foramen. Soft tissue within the foramen is dissected away, exposing the nerves to be sectioned and the radicular arteries that are preserved (only T-1 to T-4 and T-11 to L-1). The nerves are lifted slightly with a nerve hook and a clip is applied proximally to prevent the formation of a patent subarachnoid space and to prevent a cerebrospinal fluid leak. The nerve roots are then transected either by knife or by electrocautery distal to the clip.

The procedure is repeated for each level. The fascia is closed with interrupted nylon sutures, and the skin is closed in two layers. Patients are encouraged to ambulate

Table 1. Patient Characteristics

Patient No.	Sex	Age	Etiology	Nerve Roots Ablated	Length of Follow-up (wk)	Outcome
1	F	73	Squamous	T-10 to T-11	22	E
2	M	60	Mesothelioma	T-2 to T-5	19	N
3	M	50	Squamous	T-6 to T-7	13	E
4	F	43	MS/A	T-8 to T-9	31	P
5	F	71	Squamous	T-8 to T-10	6	E
6	M	60	MS/A	T-2 to T-3	30	E
7	M	52	Adenocarcinoma	T-3 to T-5	29	P
8	M	59	Adenocarcinoma	T-7 to T-8	21	E (A)
9	F	69	Adenocarcinoma	T-10 to T-12	20.5	P
10	M	67	Adenocarcinoma	T-8 to T-10	22	E
11	M	61	Adenocarcinoma	T-5 to T-8	27	E
12	M	58	Squamous	T-3 to T-4	17	E
13	M	39	Sarcoma	T-2 to T-5	20.5	P
14	F	46	Sarcoma	T-2 to T-4	45	E (A)

A = patient alive; E = excellent pain relief; F = female; M = male; MS/A = mixed squamous/adenocarcinoma; N = no pain relief; P = partial pain relief.

as soon as possible after operation and do not require special orthoses or other precautions.

Results

Patient characteristics including age, sex, etiology, length of follow-up, and results of treatment are listed in Table 1. In nine patients (64%), the procedure provided complete pain relief and a rapid decrease of need for narcotics. In 7 patients, relief lasted until death (median survival, 22 weeks), and in 2 surviving patients it lasted until the last follow-up visit, 21 and 45 weeks, respectively, after the procedure. In 4 patients (29%), after an initial pain-free period (average, 4 weeks), pain recurred but was estimated not to exceed 30% to 40% of the initial pain. Recurrence was judged to be the result of disease progression and involvement of adjacent nerve roots. Only 1 patient experienced unsatisfactory pain relief and required high-dose narcotics throughout the course of the illness. This result probably reflected a failure to identify and ablate the nerves involved. Postoperative complications included three minor nosocomial infections, which were treated successfully with antibiotics.

Comment

Pain in patients with malignant tumors of the chest cavity is usually of the somatic-visceral type. It is caused by peripheral nociceptive fiber activation secondary to the tumors invading or infiltrating nerves, soft tissue, or bone [6]. The pain is unilateral, and although it is frequently widely distributed over the chest, it projects conversionally on to one to three (rarely more) spinal nerves. Initially this type of pain responds well to analgesic and antiinflammatory drugs. However, in advanced stages that usually coincide with the exhaustion of antitumor treatments, the pain escalates acutely and becomes difficult to control by medication alone.

Specific pain procedures available to patients at this point are few, and they have various shortcomings. High cervical percutaneous cordotomy would seem to be an excellent option. However, cordotomy must be performed on the contralateral side, which often is the side of the sole functioning lung, making the operation contraindicated in the view of many [7–9]. Multilevel chemical or radiofrequency rhizotomies in the thoracic spine are cumbersome to perform because of the bony anatomy, which makes access to the neural foramen difficult [10]. It is also difficult to protect the radicular arteries, some of which are essential to the thoracic cord blood supply. Subdural or epidural neurolytics, although potentially beneficial, are difficult to gauge in terms of localization and lesion size [11, 12]. Their results, therefore, are variable, and our experience with this particular technique has been poor. Neurectomies, although potentially beneficial, present a less focused procedure. Branches (sinuvertebral and recurrent meningeal) that are involved in pain conduction from midline structures, such as the spinal elements and costochondral junction, exit the nerve at the neural foramen distal to the dorsal ganglion. A neurectomy performed distal to the foramen may skip the important proximal branches. Intrathecal infusions of opioids, although widely used in cancer patients, are less effective in patients with prior opioid tolerance, and their use raises risk of excessive depression of brainstem respiratory centers [13, 14].

Finally, open intradural rhizotomies have been regarded favorably as pain-alleviating procedures for cancer patients, particularly in the short term. However, they require multilevel laminectomies and opening of the dura mater [12, 15–19].

Therapeutic results of extrathecal rhizotomies, such as we have described here, appear to be identical with those achieved by intradural transection of the anterior and posterior roots, with the former procedure being much simpler and safer.

The factors that we consider necessary to a favorable outcome include (1) ablation of all sensory-motor and sympathetic fibers to the involved area and (2) the use of computed tomographic scan or magnetic resonance imaging to define precisely which nerve roots are involved, which eliminates the inaccuracy of selective diagnostic blockades [12].

In general, a three-level rhizotomy can be completed in less than one hour, and the procedures themselves are simple. However, the patients are debilitated individuals at high risk for virtually any operation. Judgment must be exercised on an individual basis as to whether the risk to a given patient is acceptable. Our experience is limited to patients who required four-level rhizotomies at most. It would seem unreasonable to perform this operation on a patient with a greater than four-level involvement, as it would then become a major rather than a minor procedure. Functional loss due to the procedure includes paralysis of corresponding intercostal muscles and hypoesthesia in a radicular distribution. Both sequelae are of no important consequence.

In summary, in a select group of patients with poorly controlled pain caused by malignant disease of the chest, a simplified surgical rhizotomy may provide excellent and lasting relief. The key to success lies in precise identification of the roots involved, which can be accomplished by computed tomographic scan or magnetic resonance imaging. The rhizotomy includes both the sensory and motor nerve roots, with the result that pain fibers running in the motor roots and sympathetic roots are interrupted.

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Thrombectomy of the Björk-Shiley Prosthetic Valve Revisited: Long-Term Results

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Thrombotic obstruction of the Björk-Shiley prosthetic valve is a catastrophic complication, often leading to a fatal outcome. Worldwide experience with the Björk-Shiley valve supports the need for long-term anticoagulation to prevent entrapment of the disc. Replacement of the malfunctioning device is associated with a high mortality, and therefore a more expeditious method is desirable. It is our experience that simple thrombectomy may suffice in most occasions, even for the mitral position, and especially when done through a bicameral approach in order to visualize both supravalvular and infravalvular regions. The philosophy and results with thrombectomy and disc rotation in 12 cases of thrombotic occlusion of the Björk-Shiley valve are described. It is suggested that this method may be preferable to replacement of the thrombosed prosthetic valve in select patients.

(Ann Thorac Surg 1989;48:824–8)

Thrombotic occlusion has been related to most types of mechanical valves, including the caged-ball design and tilting disc [1–3]. Progressive fibrous overgrowth usually heralds thrombus formation, and both processes result in an insidious nonreversible stenosis of the prosthetic valve orifice. Eventually, the precise mechanics of the valve are severely interfered with and acute occlusion occurs, resulting in a mortality of about 40% [4]. The incidence of this complication has been reported to be between 2% and 4% [5, 6] and, despite good results with simple thrombectomy [6–8], rethrombosis remains worrisome. As the validity of this procedure has been questioned by few [9], we reviewed our own experience with thrombectomy of the Björk-Shiley (BS) valve in an attempt to provide some insight to the dispute.

Between January 1970 and December 1987 various models of the BS valve were used in 2,092 procedures, of which thrombosis was recorded in 17 cases, for a global incidence of 0.8%. Certain aspects of the diagnosis and management of malfunction of the BS valve have been previously published [10]. Thrombectomy was carried out in 12 cases, and these experiences constitute the basis for this report.

A foul anticoagulation has been repeatedly implied in the thrombogenic process [11, 12]. However, this was observed in only 6 patients reported herein. Although the possibility of replacement exists, thrombectomy associated with disc rotation has been our method of choice in most patients, and the results are described and recommendations given for similar cases in the future.

Material and Methods

Between 1970 and 1987, the BS valve was used for valve replacement in 2,092 cases. Acute thrombosis of the prosthesis occurred in 17 cases: 13 mitral (1.2%), and 4* aortic (0.4%). Thrombectomy and disc rotation were performed in 12 (9 mitral and 3 aortic) patients, with a male/female ratio of 1:5 and a mean age of 39.7 years (see Table 1). Sinus rhythm was present in 7 patients.

Simple thrombectomy and repeated rotation of the disc were followed by generous washing and rinsing of the device through a unicameral (4 mitral and 3 aortic) or bicameral approach (5 mitral), to ensure proper disc movement and nonobstructed flow. When feasible, a rim of fibrous overgrowth was resected as well (patient 1). A conventional, stainless-steel curved sucker was employed for thrombectomy, with great care taken not to damage the struts [13]. In general, mitral cases were approached first through the left atrium, and thrombi were carefully removed from the atrial aspect of the valve. Repeated clockwise disc rotation within the sewing ring was then performed with the valve holder to facilitate full visualization of the ventricular aspect; usually, this was completed through an oblique aortic incision, gently passing the sucker through the aortic valve. No correlation could be found between valve size and interval to thrombosis, nor between valve model and location of thrombus.

A strict anticoagulation policy for all patients was implemented to attain a 25% to 35% range in prothrombin time, or a 1.4 to 1.8 quotient. Sodium warfarin (Coumadin) was used, according to patient compliance, and started when chest tubes were removed or 48 hours after weaning off bypass, whichever came first. No heparin was given in the interim, although it was used when another type of surgical procedure was to be performed, beginning 48 hours before the operation and for an equal

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* Including one inverted mitral valve in the aortic position.

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Table 1. Thrombectomy and Disc Rotation for Thrombosis of Björk-Shiley Valve

Patient No.	Sex	Age (yr)	Year of Implantation	Size (mm)	Position	Model	Anticoagulation Regimen	Adequate Anticoagulation	Comments	ECG	Implant–Reop Interval (mo)	Preoperative Studies	Approach[a]	Outcome (mo)
1	F	48	1984	25, 21	M, Ao	60°C/C	Coumadin	Yes	. . .	SR	8	Echocardiography	LA, Ao	a/w (35)
2	M	50	1977	27	Ao	Spherical	Coumadin	No	. . .	SR	20	. . .	Ao	a/w (54)
3	F	41	1978	29	M	Spherical	Coumadin	No	Herniorrhaphy	AF	8	. . .	LA, Ao	a/w (100)
4	F	48	1978	25, 23	M, Ao	Spherical	Coumadin	Yes	Hysterectomy	AF	27	. . .	LA, Ao	Died (15)
5	F	39	1972	27	M	Conical	Warfarin	Yes	. . .	AF	44	. . .	LA	a/w (150)
6	F	24	1978	25	M	Spherical	Warfarin	. . .	Pregnancy	SR	44	. . .	LA	a/w (85)
7	F	33	1973	27	Ao	Conical	AAS + DPD	. . .	TIA	SR	49	. . .	Ao	a/w (67)
8	F	29	1976	25	M	Spherical	Coumadin	Yes	. . .	SR	20	. . .	LA, Ao	a/w (128)
9	F	35	1975	25, 19, 27	M, Ao, T	Conical	Coumadin	No	. . .	AF	(48 h)	. . .	LA, Ao	Operative death
10	F	29	1974	23	Ao	Conical	AAS + DPD	. . .	Pregnancy	SR	8	Aortogram	Ao	a/w (60)
11	F	65	1980	27	M	60°C/C	Coumadin	Yes	. . .	AF	10	. . .	LA, Ao	a/w (85)
12	F	36	1978	27	M	Spherical	Coumadin	No	Embolism	SR	6	. . .	LA, Ao	a/w (67)

[a] Unicameral or bicameral.

AAS + DPD = aspirin + dipyridamole; AF = atrial fibrillation; Ao = aorta; a/w = alive and well; C/C = convexoconcave; ECG = electrocardiogram; LA = left atrium; M = mitral; SR = sinus rhythm; T = tricuspid; TIA = transient ischemic attack.

lapse of time after operation. In the early part of our series, antiaggregation with aspirin (1 g/day) and dipyridamole (300 mg/day) was used, but Coumadin became the mainstay of anticoagulation in 1975.

Results

Clinical presentation was compatible with acute pulmonary edema in 9 patients, congestive heart failure in 2 patients, and cardiogenic shock in 1 patient. All patients were rapidly evaluated in the emergency room and assessed with fluoroscopy for valve and disc performance. When clinical deterioration mandated immediate action, the patient was rushed to the operating room. Eventually, an echocardiogram could be done in patient 1. In another patient with a thrombosed aortic valve, an aortogram could be obtained and showed a stuck disc permitting a 3+ regurgitation. The interval between the first and second procedures averaged 22.1 months (48 hours to 49 months).

Thrombosis was precipitated by changes to and from oral anticoagulants and heparin in patients 3, 4, and 6 due to a herniorraphy, hysterectomy, and pregnancy occurring 2 months, 1 week, and 2 months, respectively, before the clinical diagnosis of thrombosis was made. In patient 7 a transient ischemic attack ensued five days before the valve thrombosed; and in patient 12, frequent traveling abroad resulted in a less than perfect anticoagulation.

Surgical mortality was 8.4%. The only in-hospital death occurred in a patient with multiple-valve replacement that required reoperation 48 hours later. A minimal thrombus was detected on the minor strut, and despite a successful thrombectomy, the patient fell into a coma and died on postoperative day 15. Long-term survival was 90%. A late death (patient 4) occurred 15 months after mitral thrombectomy. Autopsy was performed, and no thrombi could be found in the valve or in the coronary arteries; thus, death was attributed to a tachyarrythmia. Patient 5 underwent an uneventful ovariectomy and appendectomy 2 months after thrombectomy. In patient 6, reoperation resulted in a miscarriage. Patient 10 had undergone thrombectomy in month 5 of pregnancy, which ended with a miscarriage 2 months later. This patient underwent an uneventful hysterectomy 7 years later. All survivors improved dramatically after the thrombectomy and no case of reoccurrence has been reported to date.

Comment

Thrombotic occlusion is one of the most serious complications associated with use of mechanical valve prostheses. A high incidence was reported with the standard disc model of the BS valve [1, 2], which was later reduced with the convexoconcave model [11].

Thrombosis of the BS valve occurs gradually, and only when the stenosis becomes critical do problems appear. Uniformly, absence of the normal opening and closing sounds is documented, and little time remains for more sophisticated studies. An echocardiogram and angiogram could be done in 2 of our patients, and surgical indication was based solely on clinical grounds in the remainder. Fatal consequences may derive from a more conservative attitude as most patients are seen with congestive heart failure or acute pulmonary edema, or both. However, some reports encourage an echocardiogram to confirm the clinical diagnosis [14] and to show thick linear echoes with no discernible disc motion in the aortic position and a slow, rounded pattern of opening and closing in the mitral position.

Long-term anticoagulation has been regarded as necessary for adequate function of the BS valve and, in fact, only 1 of 7 patients described by Ben-Zvi and associates [14] had a correct level of anticoagulation when thrombosis occurred. This was also confirmed by Fernandez and colleagues [5], who found an incorrect level of anticoagulation in 3 of 7 patients with aortic thrombosis. This factor has been widely recognized to be the single most important determinant of valve thrombosis in either the aortic or mitral position, overshadowing the dubious relevance of other variables such as atrial fibrillation, previous emboli, or left atrial thrombus. In our own series, a satisfactory level of anticoagulation was achieved with sodium warfarin in patients 1, 4, 5, 8, and 11 when thrombosis occurred, and the level was incorrect in 4 other patients. It is noteworthy that surgical procedures other than cardiac ones should alert the surgeon to check the coagulant status in these patients, for serious complications may arise even when the changes with heparin are done carefully. Pregnancy is also a delicate situation, and procedures during pregnancy may lead to miscarriage, either immediately (as in patient 6) or during the postoperative period (patient 10). All in all, anticoagulation was assumed to be noneffective in 7 (58.3%) patients.

The incidence of thrombosis with older BS models ranged between 7.8% for the mitral position [15] and a prohibitive 26.8% for multiple-valve replacement [16]. This was reduced with the newer models to a more acceptable 0.46%/patient-year for all valve positions, 0.79% for single mitral valve replacement, 0.18% for the aortic position, and 0.63% for the tricuspid [17]. In our experience, a global incidence of 0.8% was found, disclosed as follows at 5- and 10-year follow-ups, respectively: 0.97% and 1.0% for the mitral position, 0.29% and 0.32% for the aortic position, and 0.63% and 0.67% for combined mitral and aortic replacement.

Both in vitro and in vivo studies [18] suggest that all clinical valves in use are stenotic, with the St. Jude Medical yielding the least pressure gradient, followed by the more restrictive Medtronic Hall and the BS valve. Reoperation of prosthetic valve obstruction is associated with a high mortality, and replacement of the thrombosed valve has resulted in a 40% mortality, as opposed to 8.3% for simple thrombectomy and disc rotation, with excellent long-term results. We do not favor extrusion of the disc [11] because such a maneuver may alter the delicate geometry of the valve and requires great expertise, whereas thorough debridement may suffice in most cases, as shown by others [6, 7, 8, 19] and our own thrombus-free cumulative survival of 946 patient-months (mean survival, 78.8 months).

One more detail deserves further comment; namely disc rotation within the sewing ring. There is no scientific

Table 2. Thrombotic Obstruction of the Björk-Shiley Valve

Author	Reference	Year	No. of Patients	Position (%) M	Position (%) Ao	Comments
Fernandez et al	5	1976	433	1.6[a]		Thrombectomy in 1 M (HM, 0%)
Byrd et al	6	1975	148	. . .	4.0[a]	Thrombectomy in 5 (HM, 0%)
Venogupal et al	8	1986	847	2.5	0.3	Overall mortality, 1.6%.
Bjork and Henze	11	1973	300	. . .	0.6	Thrombectomy in all (HM, 0%). Coumadin and DPD postop, thrombectomy and disc extrusion
Ben-Zvi et al	14	1974	121	. . .	5.0	Thrombectomy in 6 (HM, 20%)
Moreno Cabral et al	19	1978	159	5.9	2.7	Overall mortality, 4.4%. Thrombectomy in 2 Ao (HM, 0%)
Martinel et al	20	1987	2,176	0.4[b]	0.4[b]	Overall mortality; 0.1%.[b] Thrombectomy in 75% of Ao, and replacement in M.
Sutton et al	21	1978	209	6.5	4.0	First reported M thrombectomy.
Montero et al	(present report)	1987	2,092	1.2	0.4	Overall mortality, 0.8%. Thrombectomy and rotation of disc advocated and two-chamber approach.

[a] Delrin disc; [b] Incidence of reoperation for thrombotic occlusion.

Ao = aortic; DPD = dipyridamole; HM = hospital mortality; M = mitral.

basis for this rather empirical action, but when an otherwise normal prosthesis with adequate seating and a flawless implantation technique becomes thrombosed, it seems appropriate to modify the previous orientation of the valve opening, as has been our policy for these cases (Table 2).

Therefore, when thrombectomy is performed through a bicameral approach and disc rotation is associated, excellent results can be anticipated and, in our view, the risk of rethrombosis should be equal to that of the first implantation. Replacement of the thrombosed valve should be limited to cases of extensive, circular pannus underlying the thrombuslike material, or when a primary mechanical cause (ie, disc wear, mobile strut, irregular ring contour) is found.

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REVIEW OF RECENT BOOKS

Anesthesia and the Heart Patient

Edited by Fawzy G. Estafanous, MD
Boston, Butterworths, 1989
367 pp, illustrated, $65.00

Reviewed by Mark Hilberman, MD

This book evolved from a symposium of the same title held at the Cleveland Clinic in November 1986. As a symposium proceeding it is generally well written and well edited.

For the cardiac anesthesiologist interested in the field there are good brief reviews representing individual viewpoints on anesthetic drug selection and anesthetic considerations for a number of specific operative interventions. Several contributions by surgeons and internists at the Cleveland Clinic are also worthwhile. The short chapters offer quick access to relevant information and are usually well referenced.

For cardiothoracic surgeons desiring information on anesthetic drugs, attitudes, or practices affecting their patients one of several short comprehensive texts might be more suitable. Carol L. Lake's well referenced text *Cardiovascular Anesthesia* (Springer-Verlag) or Stephen J. Thomas' *Manual of Cardiac Anesthesia* (Churchill-Livingstone) come to mind.

Boulder, Colorado

Effects of Atrial Cardioplegia on the Ischemic Right Ventricle After Acute Coronary Artery Occlusion and Reperfusion

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Right atrial cardioplegia has been advocated as a simple method of delivering retrograde cardioplegia. Passive distention of the right heart inherent with right atrial cardioplegia has been shown to impair right ventricular function in a canine model of global ischemia. This study was designed to compare right ventricular performance after right atrial cardioplegia administered intermittently (n = 5) and continuously (n = 5) with coronary sinus retrograde cardioplegia (n = 5) and aortic root cardioplegia (n = 8) in a canine model of acute right ventricular ischemia and reperfusion. Right ventricular performance was assessed using the load-independent relationship of end-systolic pressure versus dimension (myocardial fiber length). Right ventricular performance was well preserved after reperfusion in those dogs protected with intermittent right atrial cardioplegia (95% of control). Results with continuous right atrial cardioplegia (66% of control) and coronary sinus retrograde cardioplegia (40% of control) demonstrated diminished postreperfusion right ventricular performance. Right ventricular performance in the group protected with aortic root cardioplegia was significantly impaired after reperfusion when compared with all retrograde groups (34% of control, $p < 0.05$). In this model, postreperfusion right ventricular performance was preserved in the right atrial cardioplegia groups despite passive ventricular distention. All methods of retrograde cardioplegia resulted in superior preservation of right ventricular performance when compared with standard aortic root cardioplegia.

(Ann Thorac Surg 1989;48:829–34)

Nonuniform distribution of cardioplegia to regional myocardium supplied by obstructed coronary arteries remains a basic obstacle to "ideal" myocardial protection during coronary artery bypass grafting (CABG) [1–3]. Retrograde cardioplegia achieves uniform distribution of cardioplegic solution throughout the left ventricle in the presence of obstructed coronary arteries [4–6]. Because the venous drainage of the right ventricle is nonhomogeneous, questions remain regarding the uniformity of cardioplegia delivery to the right ventricle and portions of the interventricular septum with coronary sinus cardioplegia (CSC) [7]. A method of retrograde right atrial cardioplegia (RAC) has been described and extensively utilized clinically [8]. Right atrial cardioplegia allows for complete perfusion of the right ventricle via the thebesian venous network. Right atrial cardioplegia does, however, produce limited passive distention of the right ventricle, raising concerns of myocardial cellular injury and postoperative right ventricular dysfunction [9].

The present study was designed to assess the efficacy of RAC when compared with CSC and standard aortic root cardioplegia (ARC) in preserving right ventricular (RV) performance. A canine model of acute right coronary artery occlusion and reperfusion was utilized to simulate prevailing conditions during revascularization in the acutely ischemic right ventricle.

Material and Methods

Twenty-three mongrel dogs (15 to 20 kg) were anesthetized with Nembutal (pentobarbital sodium; Abbott Laboratories, North Chicago, IL; 30 mg/kg), intubated, and placed on a volume cycled respirator. Polyvinyl catheters were inserted into the femoral artery and vein to monitor central aortic pressure and for infusion of fluids. Median sternotomy was performed and the heart was suspended in a pericardial cradle. The dogs were heparinized (300 mg/kg) and left femoral artery and bicaval cannulation was performed for cardiopulmonary bypass (CPB). A crystalloid pump prime was used, and systemic normothermia was maintained throughout the experiment. Systemic pH, partial pressure of carbon dioxide, and partial pressure of oxygen were monitored and adjusted as necessary.

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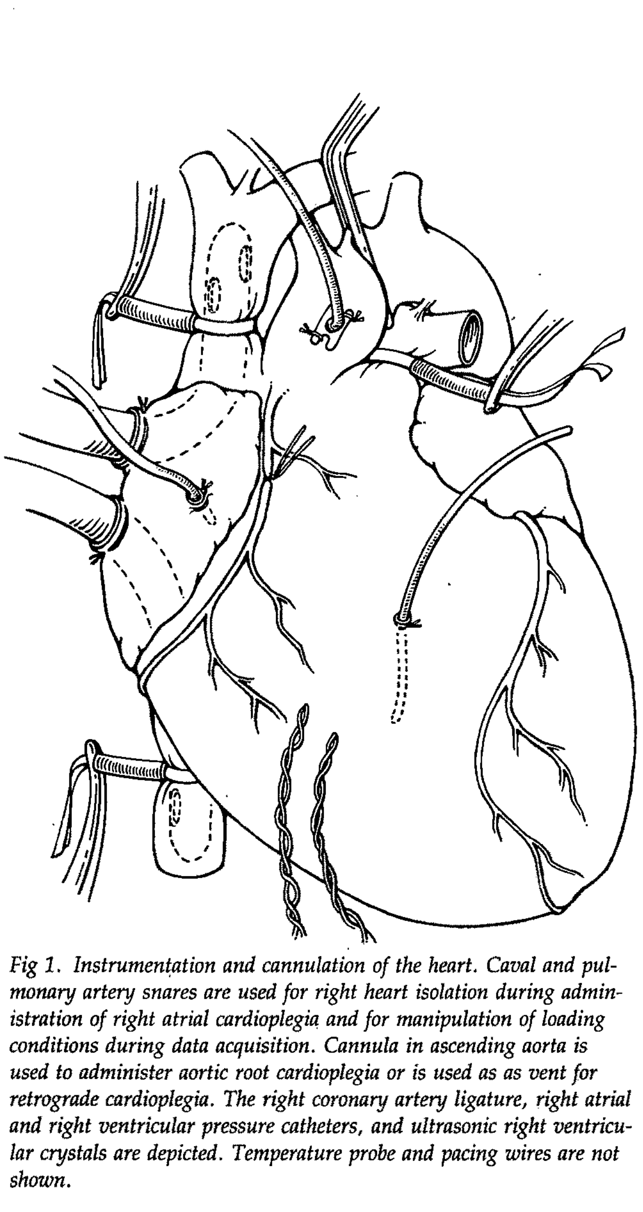


Fig 1. Instrumentation and cannulation of the heart. Caval and pulmonary artery snares are used for right heart isolation during administration of right atrial cardioplegia and for manipulation of loading conditions during data acquisition. Cannula in ascending aorta is used to administer aortic root cardioplegia or is used as as vent for retrograde cardioplegia. The right coronary artery ligature, right atrial and right ventricular pressure catheters, and ultrasonic right ventricular crystals are depicted. Temperature probe and pacing wires are not shown.

Instrumentation of the heart is illustrated in Figure 1. Umbilical tapes and snares were placed around both venae cavae to establish total CPB in all groups. Those dogs receiving atrial cardioplegia had the main pulmonary artery snared to achieve right heart isolation. A fluid-filled polyvinyl catheter was inserted into the right ventricle through the anterior RV wall to monitor RV pressure. Piezoelectric ultrasonic crystals (5 MHz, Triton Technologies, Inc, San Diego, CA) were placed in the RV myocardium 1.0 to 1.5 cm apart to measure changes in RV free wall fiber length (chord length). The ultrasonic crystals were meticulously placed at the level of the acute marginal artery and oriented parallel to the direction of the myocardial fibers. Care was taken during placement to avoid hemorrhage and development of a hematoma that might affect regional myocardial function. A myocardial thermistor (Mon-a-therm, Inc, St. Louis, MO) was placed in the RV free wall myocardium. Myocardial temperatures were recorded at baseline and every 20 minutes during CPB. All hemodynamic data were recorded on a multichannel recorder (HP 7758B, Hewlett Packard, Palo Alto, CA).

Experimental Protocol

After instrumentation of the heart and cannulation for CPB, acute RV ischemia was induced in all dogs using the method described by Connolly and colleagues [10]. A ligature was placed around the right coronary artery proximal to the acute marginal artery. Cardiopulmonary bypass was initiated after 60 minutes of ischemia or upon development of intractable ventricular arrhythmias. The development of RV ischemia was monitored during this period by two-dimensional contrast echocardiography [11, 12]. The dogs were assigned to one of the four cardioplegia groups, and global ischemia with cardioplegia was instituted for 60 minutes. Systemic normothermia was maintained throughout the bypass period, and topical hypothermia was not used.

Immediately before the release of the aortic cross-clamp, the right coronary artery ligature was released and the myocardium was reperfused for 90 minutes. The dogs were then weaned from CPB. All dogs underwent ventricular pacing at a rate of 120 bpm while off CPB.

Cardioplegia Techniques

Blood cardioplegia was used throughout the study. Crystalloid cardioplegia was diluted 1:4 with blood and delivered at 4°C (Bentley HE 100 Cardioplegia Delivery System, Bentley Labs, Inc, Irvine, CA). The initial liter of cardioplegia contained "high potassium" (120 mEq/L) and subsequent doses contained "low potassium" (60 mEq/L). These are concentrations of potassium in the crystalloid cardioplegia solution before dilution with blood (one part crystalloid to four parts blood). Topical cooling was not used. The dogs were divided into groups as detailed below.

Group 1 dogs received standard aortic root cardioplegia (ARC; n = 8) administered through an aortic cardioplegia needle (14 gauge, DLP, Inc, Grand Rapids, MI). An initial dose was given to achieve diastolic arrest and to obtain an RV temperature below 18°C. Subsequent doses were administered every 20 minutes or upon development of myocardial electrical activity during the 60 minutes of global ischemia.

Group 2 dogs received intermittent right atrial cardioplegia (IRAC; n = 5). Cardioplegia was delivered directly into the right atrium with a 7F balloon-tipped catheter; RV pressures were monitored and maintained below 40 mm Hg. Initial and subsequent doses were given according to the protocol outlined for group 1.

Group 3 was given continuous right atrial cardioplegia (CRAC; n = 5). Cardioplegia was delivered as for group 2. The initial dose was given to achieve diastolic arrest and a ventricular temperature of 18°C or below. Subsequently, cardioplegia was administered at a constant rate of 60 mL/min for 60 minutes.

Group 4 dogs received coronary sinus cardioplegia (CSC; n = 5). Delivery was achieved by direct intubation of the coronary sinus with a 7F balloon-tipped catheter through a right atriotomy. Initial and subsequent doses were administered as in groups 1 and 2.

Data Acquisition

Right ventricular temperature was measured every 20 minutes during the period of global ischemia. Right ventricular pressure and dimension analog data were acquired simultaneously at varying preload and afterload. Preload was altered by inflow (caval) occlusion, and afterload was manipulated by occlusion of the main pulmonary artery. Data were collected at baseline off CPB (before right coronary artery ligation) and after 90 minutes of reperfusion off CPB.

Utilizing the point of maximal right ventricular chord shortening as representing end systole, the slope of the end-systolic pressure–dimension relationship was derived by linear regression. This slope represents a load-independent measure of ventricular performance [13–15]. Comparisons were made within groups to assess the impact of the four methods of myocardial protection on right ventricular performance after acute RCA occlusion and reperfusion.

Animals were killed after acquisition of data. All animals were humanely treated in strict accordance with National Institutes of Health guidelines for humane animal care [16].

Statistical Methods

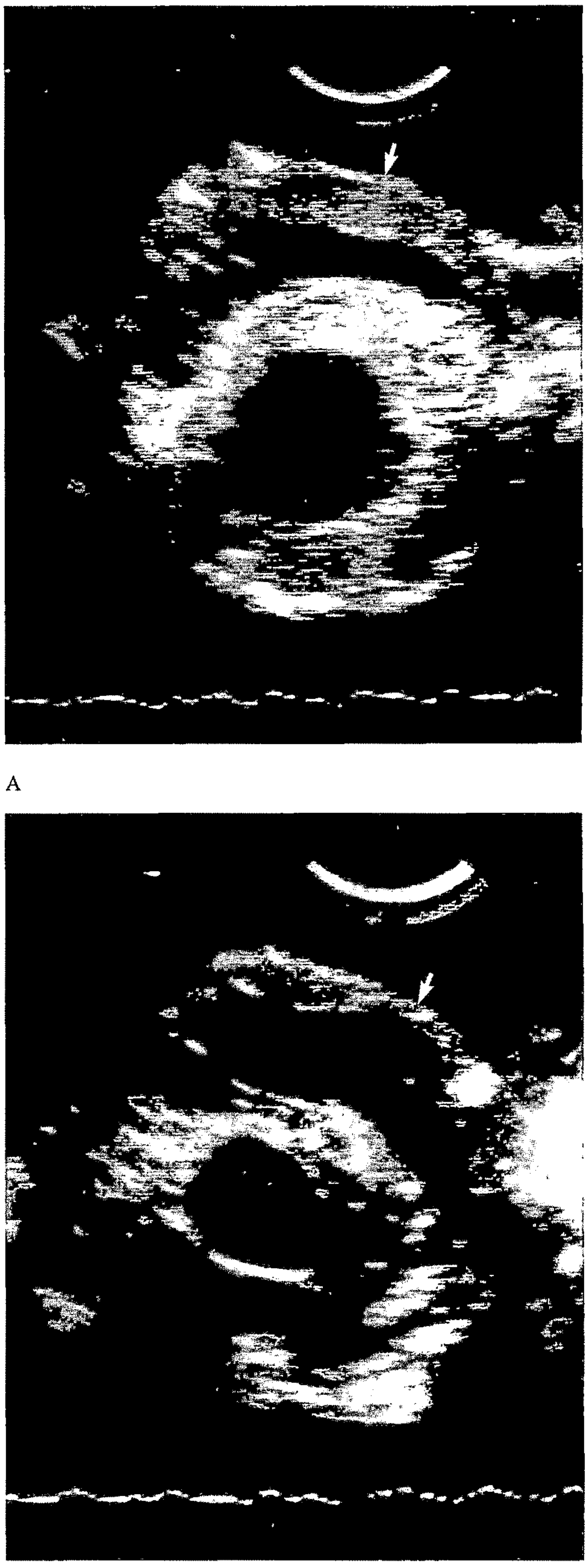
All data are presented as mean values ± the standard error of the mean. Comparisons within groups were made using the Wilcoxon signed rank test. Comparisons between groups were made using the Kruskal-Wallis one-way analysis of variance with multiple comparisons corrected by Dunn's procedure. Differences were considered statistically significant at a *p* value of less than 0.05.

Results

Right coronary artery ligation consistently and reproducibly induced RV ischemia in this model. The coronary occlusion time was less than the planned 60 minutes in all groups due to the onset of intractable ventricular arrhythmias or right heart failure. Occlusion times were similar in all groups (group 1, 37 ± 7 minutes; group 2, 39 ± 10 minutes; group 3, 38 ± 10 minutes; group 4, 42 ± 7 minutes; not significant). Two-dimensional echocardiography demonstrated wall motion abnormalities (dyskinesis and akinesis) of the RV free wall. Contrast-enhanced echocardiographic images demonstrated diminished perfusion of the RV free wall myocardium (Fig 2). Postmortem nitrotetrazolium staining performed on selected hearts demonstrated myocardial damage in the RV free wall and outflow tract that correlated with the two-dimensional echocardiograms.

Cardioplegia Requirements

The volume of cardioplegia required to achieve arrest and the total quantity of cardioplegia required to maintain arrest varied widely with the technique of administration (Table 1). Atrial cardioplegia groups 2 (IRAC) and 3 (CRAC) required significantly more cardioplegia to achieve and sustain arrest than did group 1 (ARC) and group 4 (CSC). The cardioplegia requirements for group 2 and group 3 were similar.


A

B

Fig 2. Two-dimensional contrast myocardial echocardiographic images before right coronary artery ligation (A) and 30 minutes after ligation (B). Arrows *indicate region of diminished right ventricular perfusion.*

Table 1. Comparison of the Four Cardioplegia Groups

Group	Cardioplegia Delivered (mL)	Time to Arrest (s)	Time to Spontaneous Rhythm (s)
1 (ARC)	1375 ± 99	48 ± 6	80 ± 11[a]
2 (IRAC)	3580 ± 856[b]	196 ± 38	166 ± 53[a]
3 (CRAC)	3720 ± 218[c]	197 ± 14[b]	106 ± 23[a]
4 (CSC)	1360 ± 207	297 ± 86[b]	38 ± 8

[a] $p < 0.05$ compared with group 4. [b] $p < 0.05$ compared with group 1. [c] $p < 0.05$ compared with groups 2 and 4.

ARC = aortic root cardioplegia; CRAC = continuous right atrial cardioplegia; CSC = coronary sinus cardioplegia; IRAC = intermittent right atrial cardioplegia.

Efficiency of Cardioplegia Administration

The time required to achieve arrest and the interval between cross-clamp removal and the onset of spontaneous rhythm are shown in Table 1. The time to complete arrest was significantly less for group 1 (ARC) than for groups 2 (IRAC), 3 (CRAC), and 4 (CSC). There was no difference in the time required to achieve arrest between the three retrograde groups.

The time interval between release of the aortic cross-clamp and the onset of spontaneous rhythm was significantly shorter in group 4 (CSC) and similar for groups 1 (ARC), 2 (IRAC), and 3 (CRAC).

Right Ventricular Cooling

Right ventricular cooling as measured in the RV free wall during global ischemia is depicted in Figure 3. In the absence of topical cooling, all groups demonstrated a similar initial decrease in temperature over the first five minutes of global ischemia. After initial cooling, the temperatures plateaued for each group until cross-clamp removal at 60 minutes. The mean temperature achieved between 10 and 60 minutes of global ischemia was significantly higher for group 2 (IRAC) when compared with groups 1 (ARC), 3 (CRAC), and 4 (CSC) (group 2, 26.8° ± 0.2°C versus group 1, 22.9° ± 0.2°C; group 3, 19.7° ± 0.6°C; and group 4, 22.6° ± 0.4°C; $p < 0.05$). The best cooling was seen in group 3, where mean temperatures were significantly lower than in all other groups ($p < 0.05$).

Right Ventricular Function

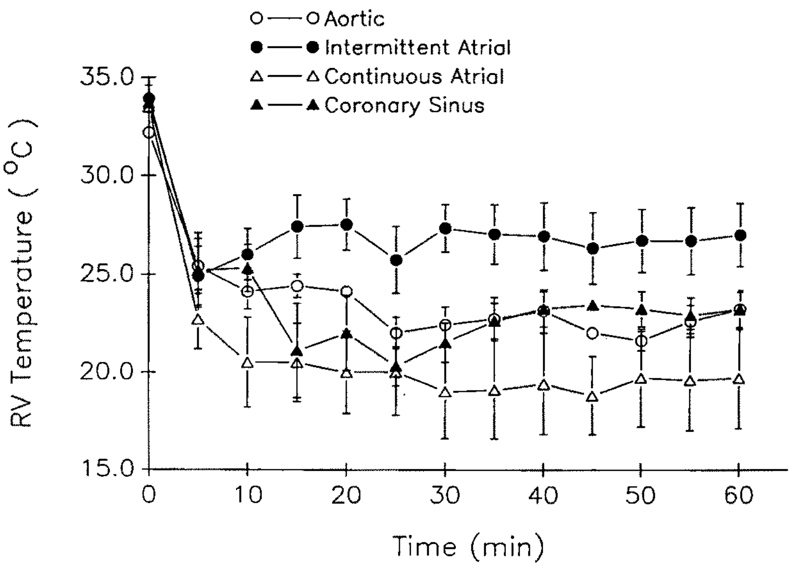
The change in the slope of the RV end-systolic pressure–dimension relationship is depicted in Figure 4. This change is measured from baseline before ligation of the right coronary artery to baseline after global ischemia and a period of reperfusion. Both sets of measurements were acquired off CPB. The slopes were similar before and after intervention for group 2 (IRAC) (18.8 ± 6.1 mm Hg/mm before versus 17.9 ± 2.7 mm Hg/mm after; NS). The slopes for group 1 (ARC) showed a significant decrease after intervention (26.2 ± 8.3 mm Hg/mm before versus 2.2 ± 6.3 mm Hg/mm after; $p < 0.05$). The slopes for groups 3 (CRAC) and 4 (CSC) demonstrated a downward trend that failed to achieve significance (group 3, 29.9 ± 7.9 mm Hg/mm before versus 19.8 ± 4.5 mm Hg/mm after; group 4, 19.8 ± 7.2 mm Hg/mm before versus 8.1 ± 4.0 mm Hg/mm after; NS).

Comment

The success of any method of myocardial protection is critically dependent on the ability to deliver cardioplegic solution to regional myocardium at risk. In patients with serious coronary artery disease, aortic root delivery of cardioplegia results in nonuniform distribution to regional myocardium supplied by stenosed or occluded coronary arteries [1–3]. The use of retrograde cardioplegia circumvents this problem for the left ventricle by delivering cardioplegia via the nonobstructed coronary venous system.

Retrograde cardioplegia may be delivered by direct cannulation of the coronary sinus or, as recently described by Fabiani and colleagues [8], via the right atrium with

Fig 3. Right ventricular (RV) temperatures (°C) during global ischemia (cross-clamp period). Intermittent atrial cardioplegia (group 2) temperatures were significantly higher than temperatures attained with aortic root (group 1), continuous atrial (group 3), and coronary sinus cardioplegia (group 4) (p < 0.05). Continuous atrial cardioplegia (group 4) temperatures were significantly lower than those seen with the other techniques (p < 0.05).



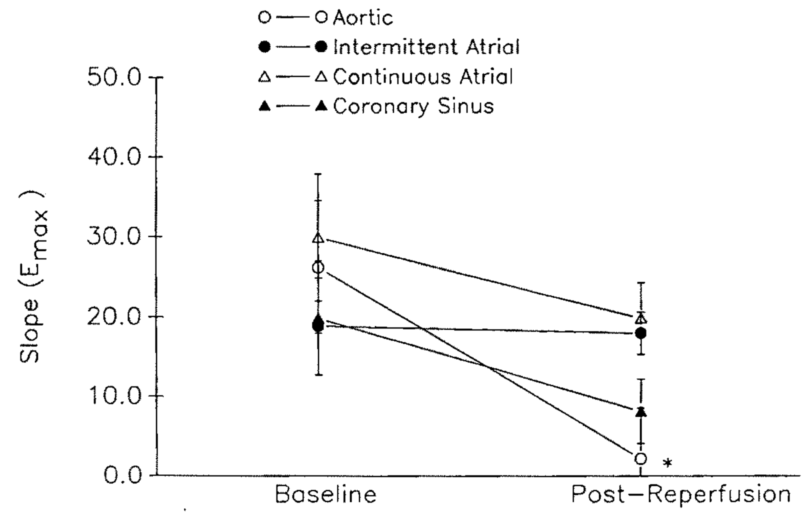


Fig 4. Changes in the slope of the end-systolic pressure–dimension relationship (E_{max}) of the right ventricle from baseline to after reperfusion. The slopes for intermittent atrial cardioplegia (group 2) demonstrate minimal variation. The decrease in the slope for aortic root cardioplegia (group 1) is significant ($p < 0.05$). Continuous atrial cardioplegia (group 3) and coronary sinus cardioplegia (group 4) slopes decrease after reperfusion but not significantly.

right heart isolation. Atrial cardioplegia obviates the need for atriotomy and cannulation of the coronary sinus with its inherent morbidity [17]. Also, there is concern over the uniformity of cardioplegia distribution throughout the right ventricle and interventricular septum with CSC. The venous drainage of the right ventricle and portions of the septum is heterogeneous. Drainage occurs through thebesian venous channels into the ventricular cavity as well as through the coronary veins and sinus [7]. Direct cannulation of the coronary sinus may also produce obstruction of proximal coronary veins by the balloon catheter [17]. Theoretically, atrial cardioplegia would provide more uniform perfusion of RV myocardium by accessing these drainage channels. Atrial cardioplegia does produce acute passive distention of the flaccid right ventricle with pressures of up to 60 mm Hg. Clinically, no adverse affects of this technique on RV function have been reported [8, 17–19]. There is, however, experimental evidence suggesting that atrial cardioplegia does impair RV performance compared with aortic root and coronary sinus cardioplegia in a canine model of global myocardial ischemia [9].

Using the load-independent relationship of RV pressure and RV chord length, we evaluated the ability of atrial cardioplegia (intermittent and continuous), CSC, and ARC to preserve RV performance. A canine model of acute RV ischemia and reperfusion was studied to mimic the clinical picture of right coronary artery occlusion and reperfusion secondary to myocardial revascularization.

Cardioplegia Requirements and Induction of Arrest

Atrial retrograde cardioplegia required significantly more cardioplegic solution than either standard ARC or CSC. This observation has been made in many of the clinical trials using RAC [17–19]. Increased cardioplegia requirements are most likely dictated by the additional volume required to fill the right heart with atrial administration. The additional cardioplegia delivered with RAC does not have an adverse effect upon the clinical outcome [17–19]. The time interval to resumption of spontaneous heartbeat was similar for groups 1 (ARC), 2 (IRAC), and 3 (CRAC). The additional cardioplegia required in the atrial groups did not impair the ability of the heart to resume spontaneous rhythm compared with group 1 (ARC) (Table 1).

All retrograde cardioplegia groups demonstrated a significant delay in the onset of arrest relative to group 1 (ARC) (Table 1). The rapid induction of cardioplegic arrest avoids an interval during which the globally ischemic ventricle is fibrillating and consuming high-energy phosphate stores. To achieve rapid diastolic arrest, an initial dose of ARC is recommended whenever any retrograde technique is used [18].

Right Ventricular Temperature

Right ventricular epicardial temperatures demonstrated similar cooling in all groups during administration of the initial dose of cardioplegia (Fig 3). Temperatures in each group then stabilized for the remainder of the cross-clamp period, with CRAC producing the best cooling and IRAC the worst. Aortic root cardioplegia and CSC delivered in the presence of right coronary artery occlusion fail to achieve adequate cooling of the RV free wall. Right atrial delivery, particularly intermittent delivery, also produces inadequate cooling despite intraventricular filling with cold cardioplegia. The RV temperatures achieved are considerably higher than those normally attained in the clinical setting. All of these techniques, when employed clinically, would benefit from adjunctive methods of cooling the right ventricle, such as topical cold slush or continuous cold saline.

Right Ventricular Performance

Right ventricular performance was assessed by using the slope of the end systolic pressure–dimension relationship as a load-independent measure of myocardial function [13–15]. In a canine model of right coronary artery occlusion and reperfusion, all methods of retrograde cardioplegia resulted in RV performance significantly superior to that achieved with ARC. Continuous atrial cardioplegia and CSC also demonstrated a downward trend in performance, which did not achieve significance. The IRAC group (group 2) showed no change from before to after reperfusion (Fig 4).

There have been reports of diminished RV function in a canine model of global ischemia with atrial cardioplegia techniques generating RV pressures of up to 60 mm Hg [9]. Clinically, atrial cardioplegia has been successfully employed for CABG without evidence of impaired postoperative RV function [17–19].

Our study was designed to approximate the clinical situation of a patient undergoing revascularization for acute ischemia due to acute right coronary artery occlusion. Under these circumstances, delivery of cardioplegia through the aortic root would only be accomplished to areas of the right ventricle supplied by patent collaterals from the left coronary system. Coronary sinus cardioplegia would allow delivery of cardioplegia to a majority of the right ventricle. However, those areas of the right ventricle and inferior septum that drain directly into the ventricle via the thebesian veins would remain unperfused. The pressures (less than 40 mm Hg) inherent with atrial cardioplegia may be outweighed by the advantage achieved by uniform distribution of cardioplegia in the ischemic right ventricle.

Further studies with this model, including assessment of ventricular metabolic variables, may help to further define the potential advantages or disadvantages of atrial delivery of cardioplegia.

Conclusions

In a canine model of acute right coronary artery occlusion with acute RV ischemia and reperfusion, we observed the following results.

1. Right ventricular cooling is inadequate with aortic root or retrograde cardioplegia techniques. The clinical use of these techniques, particularly retrograde cardioplegia, should be combined with adjunctive methods of RV cooling.
2. Atrial cardioplegia requires significantly greater volumes of cardioplegia than either aortic root or coronary sinus cardioplegia. Clinically, careful monitoring of fluid volumes and serum potassium level is necessary.
3. Retrograde cardioplegia (coronary sinus or atrial) results in a prolonged interval to diastolic arrest. Clinically, the use of these techniques should include an initial dose of antegrade cardioplegia to achieve rapid arrest.
4. Right ventricular performance is well preserved with atrial cardioplegia despite passive distention of the right ventricle.
5. Retrograde cardioplegia results in superior preservation of RV performance compared with ARC after acute right coronary artery occlusion and reperfusion.

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Simplified Method for Reoperation on the Mitral Valve

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Reoperation on the mitral valve is becoming more common because of the degeneration of bioprosthetic valves, endocarditis, and malfunction or thrombosis of mechanical valves. We advocate a technique that transforms a technically difficult operation into one that is much less tedious, time-consuming, and dangerous than reopening a sternal-split operative site the second, third, or fourth time. Favorable experience in 11 patients using right anterolateral thoracotomy without aortic or right atrial cannulation and without aortic cross-clamping or cardioplegia is presented.

(Ann Thorac Surg 1989;48:835–7)

The number of patients requiring reoperation for mitral valve disease is increasing and probably will continue to do so. The potential hazards of mitral valve operations on patients with previous heart operations include inadvertent laceration of the right ventricle and aorta, injury to patent coronary artery grafts, inadequate exposure of the mitral valve, residual trapped air in the left ventricle, and bleeding from dissection of adhesions.

As has been documented previously [1], the procedure of right anterolateral thoracotomy for reoperations on the mitral valve has been used in certain patients who have had sternal-splitting incisions initially. Exposure of the mitral valve by this method is excellent, but access to the aorta for cross-clamping and for evacuating air can be difficult. Right atrial cannulation is less secure because of previous ligation of the appendage. We now use arterial and venous cannulation of the common femoral vessels and hypothermia and ventricular fibrillation instead of cardioplegia, which allow dissection within the pericardium to be limited to exposure of the left atrium. We report results of a simplified approach to mitral valve reoperation in 11 consecutive patients operated on by four surgeons.

Material and Methods

Eight men and 3 women aged 35 to 74 years, all patients in New York Heart Association class IV and in need of mitral valve reoperation, were operated on electively with use of the following method. After each patient was placed on the operating table and a pacing Swan-Ganz catheter and an arterial monitoring cannula were inserted, anesthesia was induced and a double-lumen endotracheal tube was placed for unilateral ventilation if needed. Small folded towels were placed beneath the right scapula and the right hip with the right arm alongside the body. The skin was prepared widely and draped with steridrapes exposing chin to knees. Two saline-soaked sponges were placed beneath the steridrapes for possible future defibrillation. The right common femoral artery and vein were isolated. The skin over the fourth interspace was incised from the edge of the sternum laterally to the latissimus dorsi muscle. The intercostal muscles were divided further posteriorly for better exposure. The pericardium was opened between stay sutures anterior and parallel to the phrenic nerve at the level of the mid right atrium. Heparin was given intravenously and the right common femoral artery and vein were cannulated. The size of the arterial cannula ranged between 20 and 24F. The venous cannula (Axiom silicone femoral venous catheter; 28, 30, or 32F, 70 cm long; Axiom Medical, Inc, Paramount, CA) with multiple holes was passed from the common femoral vein into the right atrium, where it was easily felt. The venous return was attached to a centrifugal pump (Bio-Medicus centrifugal pump No. 540, Bio-Medicus, Eden Prairie, MN) that withdrew blood using a small increment of negative pressure, which provided excellent right heart decompression yielding pulmonary artery pressures that were similar to those of procedures in which right atrial cannulation, aortic cross-clamping, and cardioplegia were used.

Oxygenated blood was pumped into the femoral artery with pulsatile flow (Shiley-Stockert roller pump model 10-00-00, Shiley Inc, Irvine, CA) and the mean arterial pressure was kept at 90 mm Hg. The blood was cooled to 20°C and ventricular fibrillation occurred spontaneously. The left atrium was entered posterior to the septum, the mitral valve was replaced, and an 8F catheter was passed through the mitral valve to the left ventricular apex providing left ventricular venting and decompression. This catheter was held at the atriotomy site with a separate pursestring suture and the atrium was closed. Two hundred milligrams of lidocaine was given and the blood was rewarmed. Air was eliminated from the heart by allowing the ventricle to fill, by rotating the table from side to side while ventilating the lung, and by compressing the anterior chest wall with the patient in deep

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Trendelenberg position. The heart was electrically defibrillated, if required, with sterile external paddles. The operating table was restored to normal position. A pool of blood contained by the pericardium was allowed to accumulate above the atriotomy closure and vent sites. When good cardiac function resumed, the left ventricular vent was removed and the pursestring suture was tied. The pericardial edges were approximated. Chest tubes were placed in the right pleural space, and the chest wall was closed in layers.

Results

Ten of 11 patients survived operation, were improved, and left the hospital in satisfactory condition. In none of our 11 patients has an intraaortic balloon pump or other method of mechanical support been required. No patient was returned to the operating room for postoperative hemorrhage. No neurological injury occurred. One death occurred that was not the result of the operative technique.

Comment

Right anterolateral thoracotomy without aortic cross-clamping and right atrial cannulation is elected in certain patients for reoperation on the mitral valve because it avoids the potential for injury to the aorta, right ventricle, and coronary bypass grafts. It provides excellent exposure, involves a smaller incision with less dissection than midline sternotomy, and is superior to left posterolateral thoracotomy because it affords palpatory confirmation of the location of the tip of the venous cannula. As time progresses, we will be seeing more patients with a patent internal mammary artery graft who may require mitral valve repair or replacement in whom, with our method, dissection and control is not necessary and injury to the internal mammary artery is avoided.

Left posterolateral thoracotomy is considered for the second reoperation for mitral valve replacement, assuming median sternotomy and a right thoracotomy had been performed previously. We also recommend left thoracotomy if bypass grafts are required on the left side of the heart. Each of the lateral thoracotomy approaches is recommended as timesaving, expeditious, and safer for emergency operations for malfunctioning or thrombosed mitral prostheses.

Unexpected serious aortic regurgitation would interfere with adequacy of pulsatile aortic pressure, organ perfusion, and visibility in the operative field. To preclude such surprises, aortic root injection is performed at the time of cardiac catheterization and coronary angiography. If we are unable to perform aortic root injection, we use the Doppler echocardiography to assess aortic insufficiency. Even mild (1+) aortic regurgitation would discourage right and left thoracotomy approaches to reoperative mitral valve replacement. Finding unmanageable aortic regurgitation during an emergency operation through either lateral thoracotomy could require additional dissection to expose and control the proximal ascending aorta. We have used the Bio-Medicus pump rather than gravity venous drainage to attempt to achieve better regulatory control of venous outflow. This pump allows regulation of venous drainage by adjusting negative pressure. No difficulty has been experienced in advancing the long venous cannula. Inadvertent retrohepatic location of the tip of the venous cannula might effect inadequate venous outflow, allowing right heart distention interfering with exposure, but such complications have not occurred. Venous diversion, exposure, and visibility have been excellent in all cases.

Maintenance of systolic aortic-arterial pressure at 90 mm Hg usually requires adjusting flow rates between 1.8 to 3.0 L/min/m^2 and occasional use of Neo-Synephrine. The ideal situation is a still, small heart without excess blood in the field and avoidance of anatomical, chemical, or functional myocardial damage. We have chosen ventricular fibrillation (VF) without aortic cross-clamping as our procedure for re-replacing the mitral valve. We maintain five conditions while the heart is fibrillating: systemic hypothermia, pulsatile perfusion, mean arterial pressure greater than 90 mm Hg, spontaneous fibrillation, and decompression of the left ventricle.

There have been multiple studies on the safety of spontaneous VF. Hottenrott and co-workers [2], comparing hypertrophied and nonhypertrophied hearts, found that there were no histochemical, metabolic, or functional changes in nonhypertrophied hearts after 60 minutes of VF. Cox and associates [3] also found that VF caused no deleterious effects in nonhypertrophied hearts. They stressed the importance of maintaining the aortic pressure between 70 and 100 mm Hg. Hottenrott and colleagues [4, 5] compared spontaneous VF with electrically induced VF in dogs and found that oxygen delivery to the left ventricle was markedly reduced and that coronary flow was redistributed away from the subendocardium during electrically induced VF, but not in spontaneously fibrillating hearts.

Vinas and associates [6] determined that hypothermia does protect the myocardium in VF. Myocardial oxygen consumption and myocardial lactate production increased significantly in the normothermic group compared with the hypothermic group. Lactate extraction remained stable in the hypothermic group but increased in normothermic fibrillating hearts.

Akins [7–9] reported excellent results in groups of patients who successfully underwent elective or emergency coronary artery bypass procedures using hypothermic fibrillation. Habal and colleagues [10] compared VF in patients with and without pulsatile flow. There was anaerobic metabolism in myocardium perfused with nonpulsatile flow. The addition of pulsatile aortic flow to coronary perfusion during two hours of VF resulted in reversion to aerobic metabolism, much higher lactate extraction, improvement in metabolic function, normal left ventricular compliance, and reduction in ischemic subendocardial histological changes.

It is conceivable that defibrillation will be difficult and delayed in occasional patients.

Exposure of the mitral valve by anterolateral thoracotomy is not new. Familiarity and ease with its use are easily achieved. Silicone catheters, controlled-aspiration venous pumps, modulated pulsatile arterial pumps, and the experimentally learned and proved merits of the five conditions of cardiopulmonary bypass add up to new skills for established and recently minted cardiac surgeons alike.

In conclusion, we recommend right anterolateral thoracotomy using femoral cannulation and no aortic cross-clamping in situations in which difficult dissection of the heart is anticipated, calcified aorta is suspected, or patent saphenous bypass grafts or an internal mammary artery graft is present. Contraindications to this procedure are aortic regurgitation, severe ventricular hypertrophy, and advanced peripheral vascular disease. The rules that must be observed are the use of systemic hypothermia, pulsatile perfusion, mean arterial pressure greater than 90 mm Hg, spontaneous fibrillation, and decompression and venting of the left ventricle. This procedure does not require aortic dissection or cross-clamping with their attendant dangers, and it is safe and rapid. In cases of extreme emergency in which the prosthetic valve is acutely thrombosed or develops acute regurgitation and the patient may be in cardiogenic shock, this procedure enables the surgeon to institute cardiopulmonary bypass without delay.

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Large Animal Model of Left Ventricular Aneurysm

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In 28 Dorsett sheep, ligation of the distal homonymous (equivalent to human left anterior descending) and second diagonal coronary arteries produced a constant transmural infarct of 22.9% ± 2.5% (mean ± standard deviation) of the left ventricular mass. Serial left ventriculograms showed that within four hours the infarct segment expands, wall thickness decreases, and aneurysmal dilatation occurs and progresses over the next 60 days in all sheep. Epicardial ventricular point references indicated that adjacent noninfarcted myocardium participates in the formation of the aneurysm. Anatomy of the coronary vasculature was studied in 22 excised sheep hearts. In sheep, coronary arterial anatomy is remarkably constant. The left coronary artery provides all of the blood supply to the left ventricle and septum and only a small rim of both the anterior and posterior right ventricles. Cardiac veins from the left ventricle drain into the coronary sinus, which also receives the left azygos vein. Right ventricular veins drain separately. The essentially separate coronary circulations to the two ventricles, the paucity of coronary collateral circulation, and the consistent evolution of left ventricular infarcts into aneurysms are important advantages of the ovine model for both metabolic and ventricular mechanical studies of acute myocardial infarction and left ventricular aneurysm.

(Ann Thorac Surg 1989;48:838–45)

Approximately 1.5 million Americans suffer acute myocardial infarction each year, and left ventricular aneurysm develops in 10% to 30% [1–5]. Eighty-five percent of aneurysms are apical and result from occlusion of the left anterior descending coronary artery [4–6].

Apical aneurysms occasionally develop in dogs after ligation of the left anterior descending coronary artery, but formation is unpredictable and inconsistent [7, 8]. Moreover, the canine model is limited for studies of ischemic heart disease because of abundant natural subepicardial collateral vessels [9–11]. Not surprisingly, the coronary circulation of primates is similar to that of humans [12–14], but these animals are expensive and generally unavailable. The coronary circulation of pigs most resembles humans of all large nonprimates [10, 15–17], but these animals are difficult subjects for cardiopulmonary bypass and are unsuitable for chronic studies involving mechanical circulatory assistance. The proximal septum of the bovine coronary circulation is supplied by a branch of the right coronary artery and the remainder by branches of all three coronary vessels [18]. Furthermore, the rapid growth rate of calves impairs chronic studies. Sheep, however, are inexpensive, widely available, large, and docile; they have been bred to constant and reproducible standards and generally withstand operation well.

This study describes the coronary circulation of sheep relevant to studies of myocardial metabolism, mechanics, and ischemia and presents a method to reproducibly induce apical left ventricular aneurysms.

Material and Methods

Coronary Anatomy

Twenty-two sheep hearts were obtained from freshly killed and exsanguinated animals at a local slaughterhouse. Fresh hearts were flushed with heparinized saline (10 U/mL) and refrigerated until studied. The proximal right, homonymous, and circumflex coronary arteries of 16 hearts were dissected out and separately cannulated through the aortic root; cannulas were secured with ligatures. Multicolored injections of Batson's No. 17 polymer (Polysciences Inc, Warrington, PA) were made as described by Wolfe [19]. After arterial injections, cannulas were withdrawn and the aortic root was filled with polymer to complete the cast. After the polymer had cured for 24 hours, cardiac tissue was digested away with a saturated solution of potassium hydroxide.

For venous studies, the right atrium of six hearts was opened to suture the ostium of the coronary sinus closed. A cannula was inserted into the left azygos vein. Polymer (Batson No. 17) was injected through the cannula to produce a cast of the venous system, which was cured and cleaned as described in the previous paragraph.

Left Ventricular Aneurysm

Twenty-eight Dorsett sheep (22 to 30 kg) were anesthetized with sodium pentothal (0.5 mL/kg normal body weight), intubated, and ventilated at 18 mL/kg on a Harvard volume ventilator with an oxygen/halothane mixture in compliance with National Institutes of Health Publication No. 85-23 as revised in 1985. The surface electrocardiogram and arterial pressure from an 8.5F catheter in the left carotid artery were monitored. The chest was entered through a left fifth interspace thoracotomy and the pericardium was opened. In all sheep,

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monofilament ligatures were passed around the homonymous and second diagonal coronary arteries at a point 40% of the distance from apex to base. These vessels were ligated immediately in 19 sheep. Before ligation, each sheep received bretylium (5 mg/kg IV) and lidocaine (75 mg IV bolus and 1 to 2 mg/min infusion). In 9 sheep, eight radiopaque markers were sutured to the epicardial surface of the left ventricle in a single plane to facilitate interpretation of postoperative ventriculograms. In these sheep preinfarction ventriculograms were obtained after the animals awoke. The coronary arteries were ligated three days later through the anterior portion of the incision. After thoracotomy, a 28F chest tube was placed and the wound was closed in layers. Chest tubes were removed one to four hours after operation when drainage stopped. Animals were allowed a regular diet and activity when awake. During operation and daily for three days, all animals received chloramphenicol (2 g/IV). The carotid arterial catheter was filled with heparin-saline solution and capped.

Eight sheep were killed four to 72 hours after infarction to determine the size of the infarct. For euthanasia, animals received thiopental (0.5 mL/kg IV) and heparin (2 mg/kg IV) followed by rapid injection of 80 mEq KCl. The area of infarction was determined by differential staining of viable myocardium by triphenyl tetrazolium chloride. Triphenyl tetrazolium chloride (500 mL of 2% solution) was infused intravenously for 30 minutes while the circulation was maintained by internal cardiac compression. The heart was excised, the atria and right ventricle were removed, and the left ventricle was sectioned at 5-mm intervals. Tracings of all slices and the extent of triphenyl tetrazolium chloride staining were made onto clear acetate and all sections were preserved in formalin. Extent of the infarction was determined by planimetry (No. 9826 digitizing pad; Hewlett-Packard, Palo Alto, CA) of the total traced areas of stained and unstained myocardium. Representative histological sections were obtained from 4 sheep that were killed 3, 7, 21, and 60 days after myocardial infarction.

Serial left ventriculograms were performed in 14 sheep, 9 of which had epicardial markers. Animals were awake and standing in a harness during the study. A 7F pigtail catheter was introduced into the left carotid arterial cannula and passed retrograde into the left ventricle under fluoroscopic guidance (Model 45-162730G1, General Electric Inc, Fairfield, CT). The image intensifier was placed over the right side of the back and the cathode tube was placed below the left anterior chest (right posterior oblique projection). Images were recorded on videocassette tape after rapid injection (Model PI-10150S, Cook Inc, Bloomington, IN) of 30 mL of contrast material (Hypaque 76, Winthrop Pharmaceuticals, New York, NY). Ventriculograms were obtained before coronary ligation in the sheep with epicardial markers and, depending on the longevity of the sheep, at four hours and 3, 7, 21, and 60 days after ligation.

Changes in four axes across the ventricular chamber and changes in left ventricular wall thickness at the seven points indicated by epicardial markers were measured in 31 right posterior oblique ventriculograms (7 sheep) during systole (Fig 1). Measurements were made on projected ventriculograms and distances were corrected to millimeters by multiplying measured distances by a magnification factor. This factor was calculated for each projected ventriculogram by dividing the measured projected distance across the large (6.35 mm) epicardial markers by 6.35 mm.

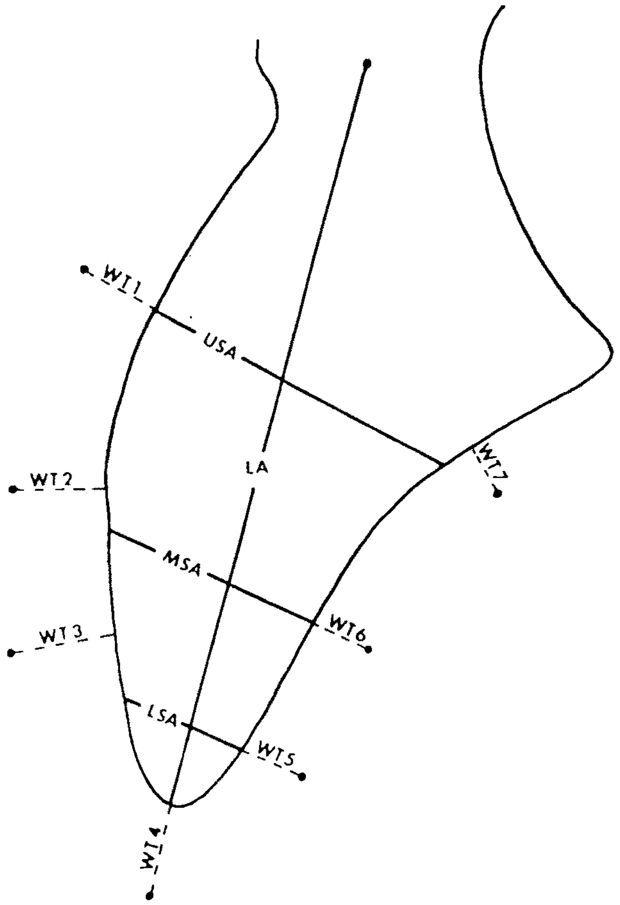

Fig 1. *Ovine left ventricle in the right posterior oblique projection.* Dots *indicate the position of the eight epicardial markers. The ventricular outline indicates the endocardial surface. Locations* WT2 *through* WT5 *were within the infarcted vascular bed of the ligated arteries. The ligatures were tied just cephalad to the* WT2 *marker.* (LA = *long axis of ventricular cavity;* LSA = *lower short axis;* MSA = *mid-short axis;* USA = *upper short axis of ventricular cavity;* WT1–7 = *wall thickness at each location of epicardial markers.)*

Results

Coronary Anatomy

Casts of injected coronary arteries show remarkably consistent coronary arterial anatomy. The left main coronary artery divides within 7.8 ± 2.0 mm (mean ± standard deviation; n = 7) into the homonymous branch, which is equivalent to the human anterior descending branch [20] (Figs 2A, 2B) and the circumflex coronary artery. The first branch of the homonymous branch originates high and branches on the anterior wall of the left ventricle. The second branch has a variable site of origin from the mid-anterior descending artery and distributes over the

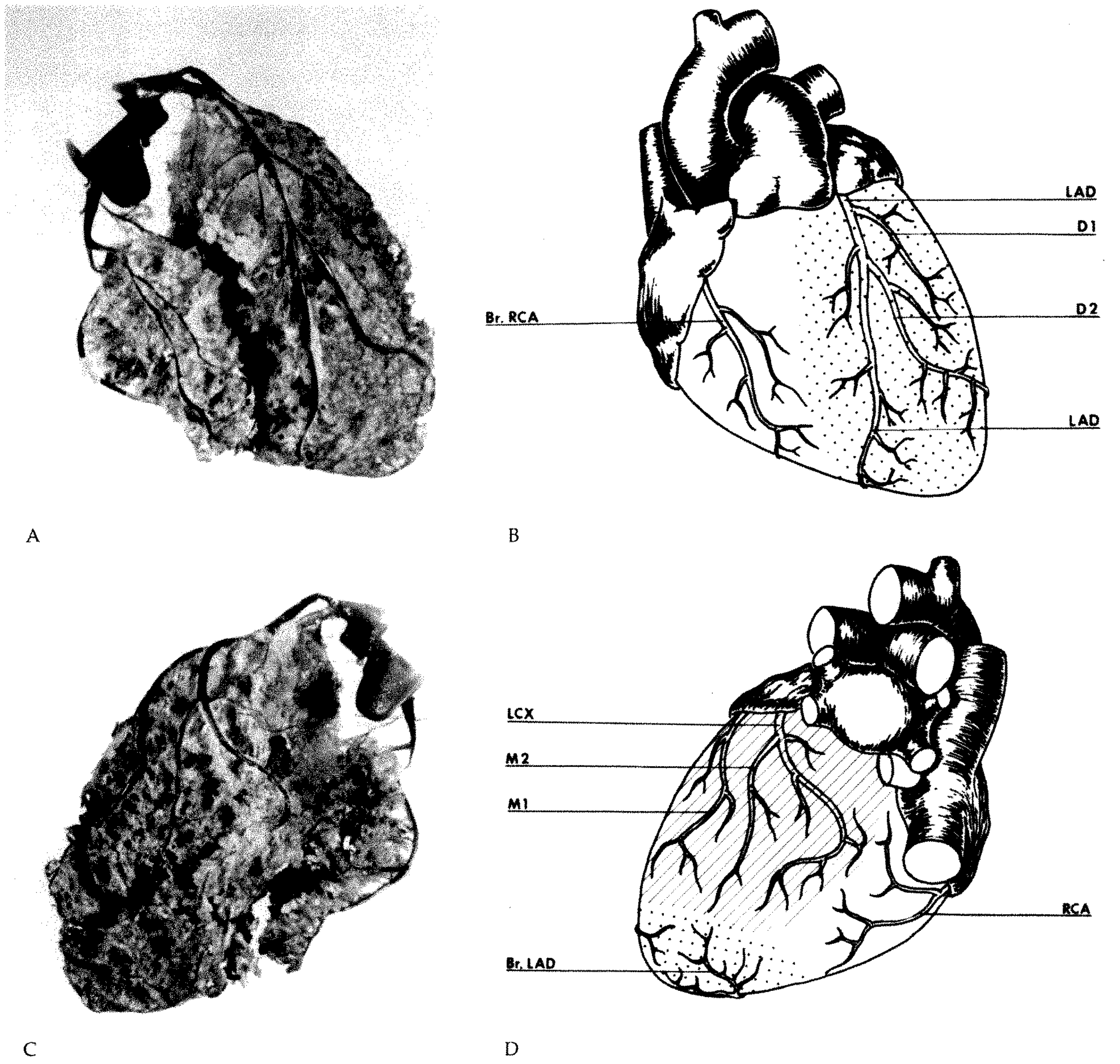

*Fig 2. Arterial casts and diagrams of the ovine heart. (A) Anterior view of a polymer cast of a heart after separate colored injections of the right, left circumflex, and homonymous arteries. (B) Diagram of the multicolored cast shown in (A). (*Br. RCA = *branch of right coronary artery.* LAD = *homonymous branch of the left coronary artery, which is equivalent to the human left anterior descending coronary artery. The first and second branches are labeled* D1 *and* D2. Stippled area *depicts the flow distribution of the* LAD *injection.* Clear area *depicts the flow distribution of the right coronary artery [*RCA*] injection.) (C) Posterior view of the same polymer cast shown in (A). (D) Diagram of the multicolored cast shown in (C). (*Br. LAD = *apical branches of the* LAD; LCx = *circumflex branch of the left coronary artery with marginal branches labeled* M1 *and* M2; RCA = *right coronary artery.* Cross-hatched *area represents the flow distribution of the LCx. The* stippled *and* clear *areas depict the distributions of the LAD and RCA, respectively.)*

lower anterior wall and apex. The terminus of the homonymous and multiple small side branches project around the apex, the anterior two thirds of the septum, and the junction of the right ventricular free wall with the anterior wall of the left ventricle (Fig 2).

The circumflex artery supplies the remainder of the left ventricle and extends around to the posterior wall to give off two to three marginal branches and a short posterior descending vessel with septal branches in the posterior interventricular sulcus (Figs 2C, 2D). The circumflex supplies the posterior septum, the inferior and lateral walls, and the junction of the right ventricle with the posterior and right walls of the left ventricle.

The right coronary artery gives off multiple small branches to the right ventricular free wall (Fig 2).

This pattern of distribution was similar in all sheep studied. The posterior descending artery never originated from the right coronary artery. The left ventricle including the septum always received its entire blood supply from the left coronary arteries. The right ventricle received

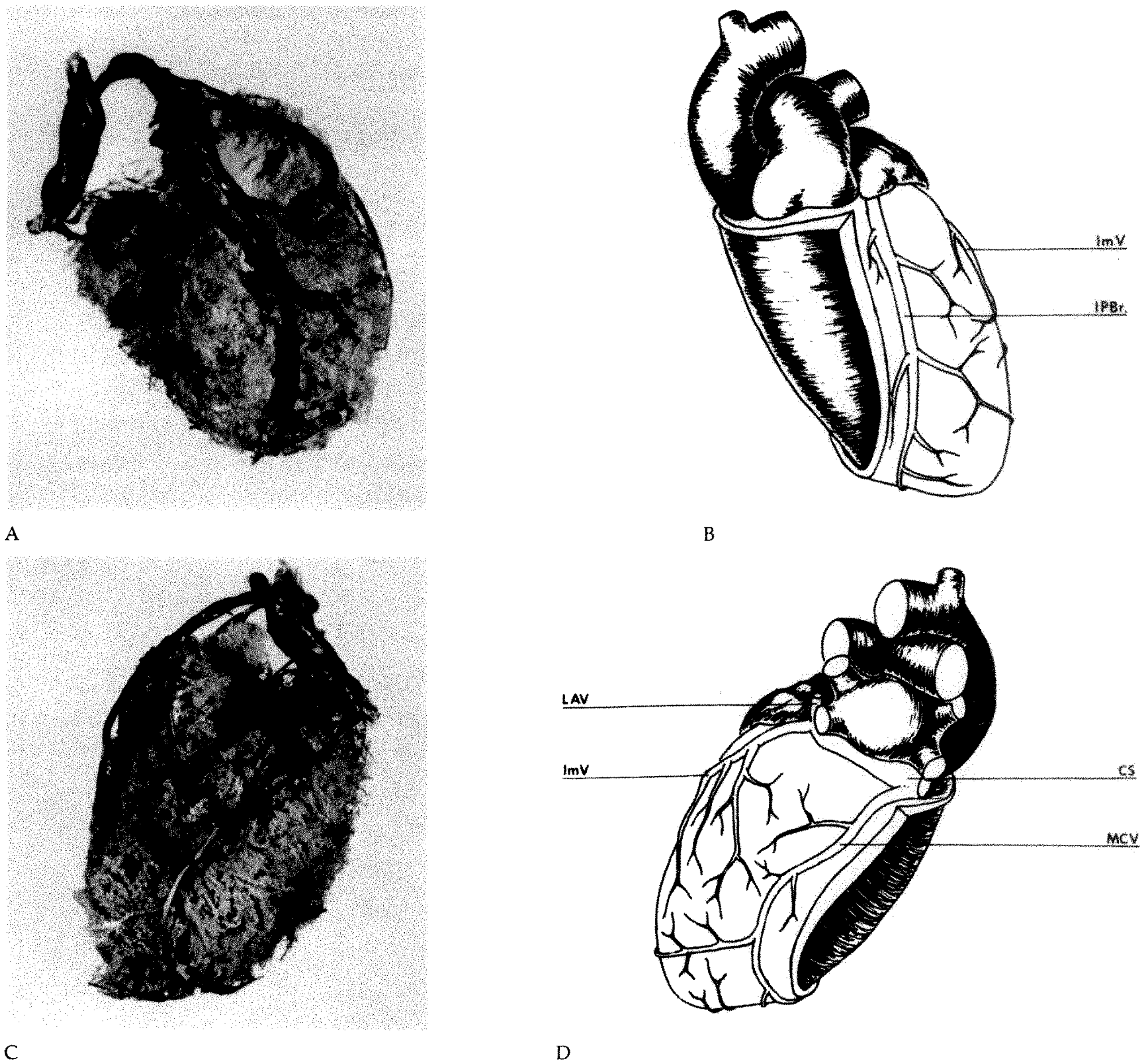

Fig 3. Polymer cast from an injection into the coronary sinus of an ovine heart. (A) Anterior view of the venous cast. (B) Diagram of the cast shown in (A). (ImV = *intermediate branch of the great cardiac vein;* IPBr. = *interventricular paraconal branch of the great cardiac vein.) (C) Posterior view of the venous cast shown in (A). (D) Diagram of the cast shown in (C).* (Cs = *coronary sinus;* LAV = *left azygos vein;* MCV = *middle cardiac vein.)*

nearly all of its blood from the right coronary artery. At the anatomical junction of the two ventricles near the apex, the left system supplied a tiny area of right ventricle.

The zone of distribution for each artery was clearly demarcated, and we found no macroscopic evidence of collateral vessels between distributions of the major or branch coronary arteries.

Three major veins that are linked by an intermeshing venous network drain into the coronary sinus and provide the principal venous drainage of the left ventricle [21]. The interventricular paraconal branch of the great cardiac vein drains the anterior wall of the left ventricle and the edge of the right ventricle (Figs 3A, 3B). The great cardiac vein joins the left azygos vein and continues as the coronary sinus into the left side of the right atrium. The intermediate branch of the great cardiac vein drains the lateral left ventricle and joins the coronary sinus at the junction of the great cardiac vein and left azygos vein (Figs 3C, 3D). The middle cardiac vein drains the postero-inferior wall and septum and drains into the coronary sinus at its junction with the right atrium. Thus the venous drainage from most of the left ventricle appears to be through the coronary sinus. The superior septum drains venous blood by an alternate route. The right ventricular venous network does not drain into the coronary sinus system. According to Ghoshal [21], right ventricular veins drain directly into the right atrium.

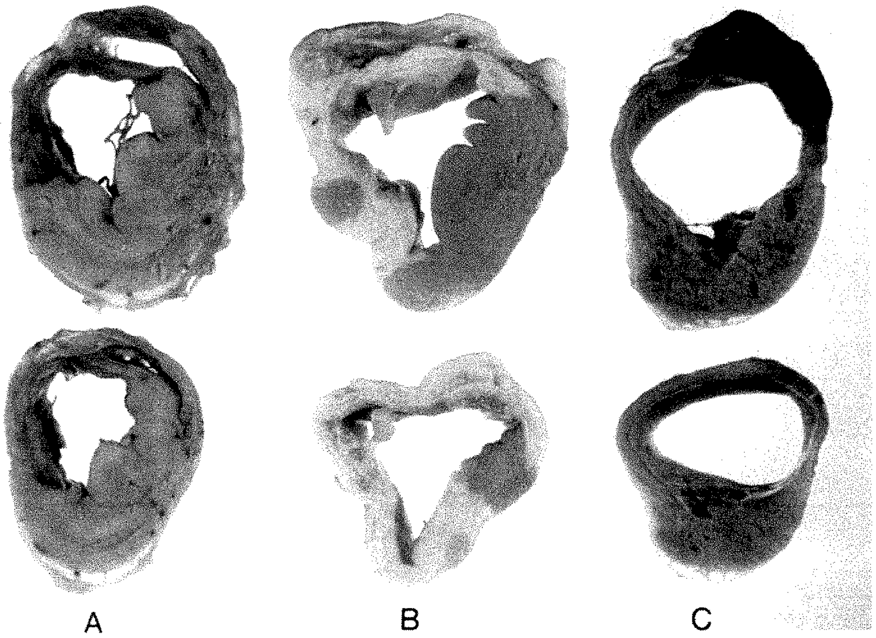

Fig 4. Ventricular cross-sectional slices of ovine hearts stored in formalin. Animals were killed 1 week (A), 3 weeks (B), and 3 months after coronary arterial ligation (C). Note the progressive thinning and expansion of the anteroseptal portion of the ventricle. Specimen (B) was stained with triphenyl tetrazolium chloride in vivo.

Left Ventricular Aneurysm

Three sheep died in the perioperative period; 8 were killed before 72 hours to determine infarct size and 17 developed evidence of left ventricular aneurysm and were killed 72 hours to 6 months after ligation of the coronary arteries.

The amount of infarcted left ventricle was determined in 8 sheep killed up to 72 hours after operation. A mean of 22.9% (standard deviation, 2.5%) of the left ventricle had transmural infarction. An adjacent small area of nonuniform partial thickness infarction was occasionally noted.

All 17 sheep that survived 72 hours or more had aneurysmal dilatation of the apex and anterolateral wall at postmortem examination. All specimens had a thin rim of viable myocardium lining the infarct. Grossly, thinning of the infarcted region was observed at three days (n = 2) with both progressive thinning and expansion of the thinned area at 1 week (n = 4) (Fig 4), 3 weeks (n = 5), 2 months (n = 1), 3 months (n = 2) (Figs 4, 5), 4 months (n = 1), and 6 months (n = 2). In the two 6-month specimens, approximately 50% to 60% of the left ventricle was aneurysmal and had a wall thickness of 1.0 to 1.5 mm. There was little change in aneurysmal wall thickness between 2 and 6 months.

Microscopically, the specimens at three days showed transmural replacement of the myocardium with focal granulation tissue (Fig 6A). Increased fibrosis and decreased cellularity was noted at 1 week (Fig 6B), and by 3 weeks (Fig 6C) the area was entirely fibrotic. Microcalcification was sometimes noted at junctions with normal myocardium (Fig 6D). At 2 months (Fig 6E) only increased thinning was noted. No calcification was noted in the 2-month specimen.

Aneurysmal dilatation, defined as an area of akinesis or

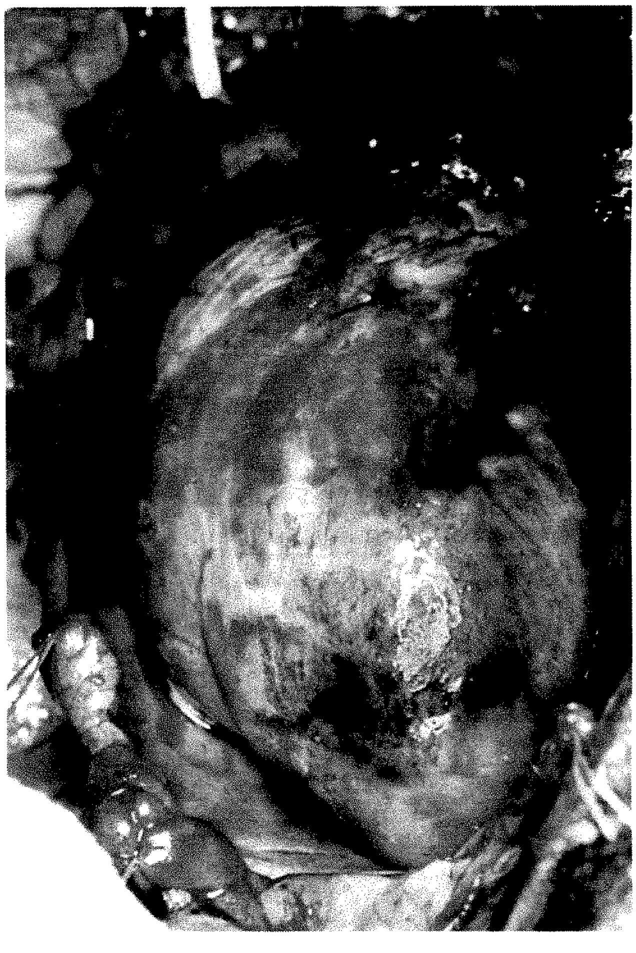

Fig 5. Operative photograph of an ovine apical left ventricular aneurysm 3 months after ligation of the coronary arteries. The animal's head is toward the top.

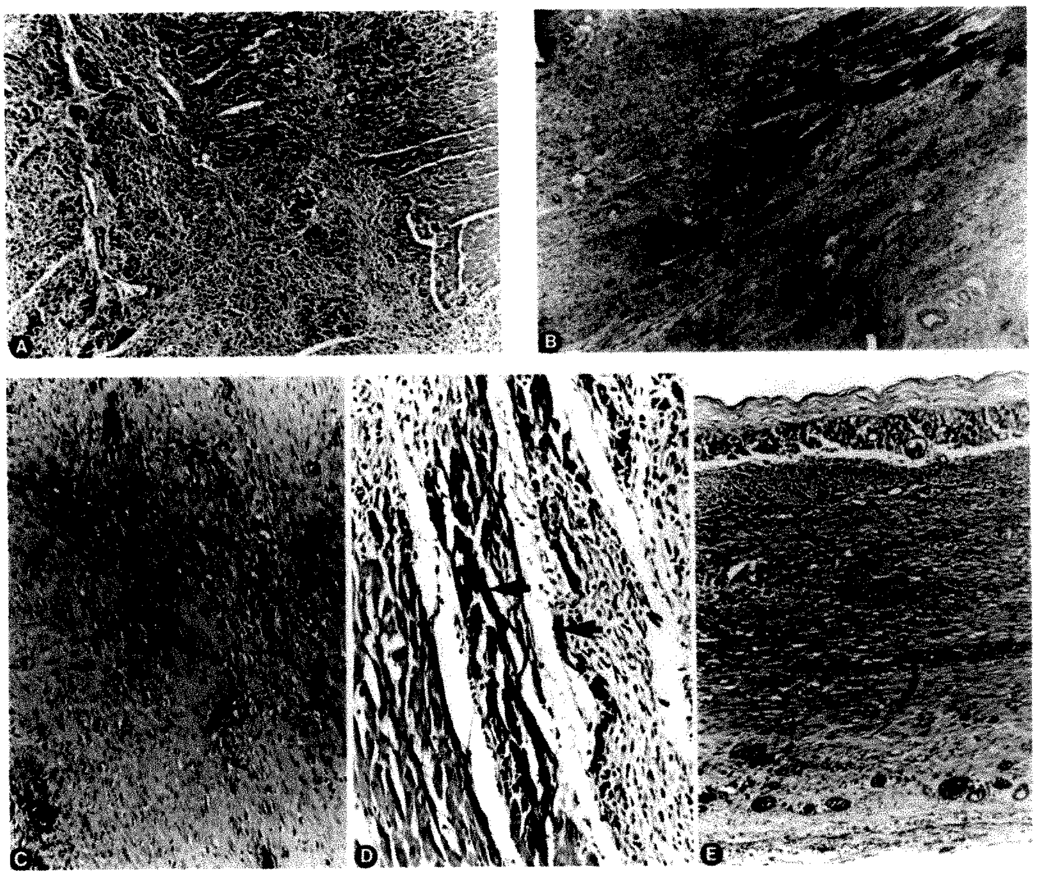

Fig 6. Histological changes noted at different time intervals after infarction. In (A), focal replacement of the myocardium with granulation tissue is noted at three days. At 1 week (B), there is decreased cellularity and increasing fibrosis, whereas at 3 weeks (C), the area is entirely fibrotic with microcalcifications (D) (arrows)*. Note thinning of the wall at 2 months (E). (A, ×100; B, ×40; C, ×100; D, ×200; E, ×40.)*

dyskinesis that involved 20% or more of the left ventricular border (right posterior oblique projection) and that was clearly surrounded by normally contracting myocardium, was observed in all 17 ventriculograms obtained three days or more after infarction (Fig 7, Table 1). Within four hours, systolic wall thickness in the area of the infarct had decreased and the diameter of the ventricle across the infarcted axis had increased (Table 1). Most of the thinning and bulging of the infarct area occurred within three days (Table 1, Fig 7). The border of the infarct was initially indistinct but became clearer by three to seven days. At 2 months, the heart was greatly enlarged and the contractile regions were limited to the anterior and posterior segments closest to the base of the heart. Adjacent, previously contractile muscle had become thinner and pulled into the enlarging aneurysm (Table 1). Mural thrombi were not observed although pooling and delayed clearance of contrast medium in the infarcted region was striking in all ventriculograms.

Comment

The anatomy of the coronary arterial circulation of sheep is remarkably consistent and offers important advantages for study of myocardial metabolism and mechanics in normal and pathological hearts. As shown by previous studies [10, 18, 20–23] and this one, the arterial circulation of the ovine left ventricle is exclusively supplied by the left main coronary artery and its tributaries; the right coronary artery makes no contribution. Only a small rim of the right ventricle near the junction of the left ventricle receives blood from the left system. Thus the right and left ventricles have separate circulations supplied by separate coronary vessels with minimal overlap. Ovine coronary vasculature facilitates metabolic measurements because the left main coronary artery supplies all of the left ventricle and little else. Furthermore, cannulation of the left azygos vein, which exclusively drains the left ventricle, facilitates collection of blood samples from the coronary sinus.

Consistent coronary anatomy permits predictable myocardial infarcts with small standard deviation. Furthermore, sheep do not have preformed coronary arterial collateral vessels [24], and macroscopic collaterals do not develop in them in response to ischemia. Schaper [10] was unable to identify collaterals in nonischemic sheep hearts or in areas of chronic ischemia induced by ameroid constriction. Hegasi [25] studied sheep, oxen, and goats and found arteriovenous and arterioarterial anastomoses only in goats. Hadziselimovic and associates [26] found that ovine intraarterial anastomoses were much more prevalent than arterioarterial anastomoses in corrosion

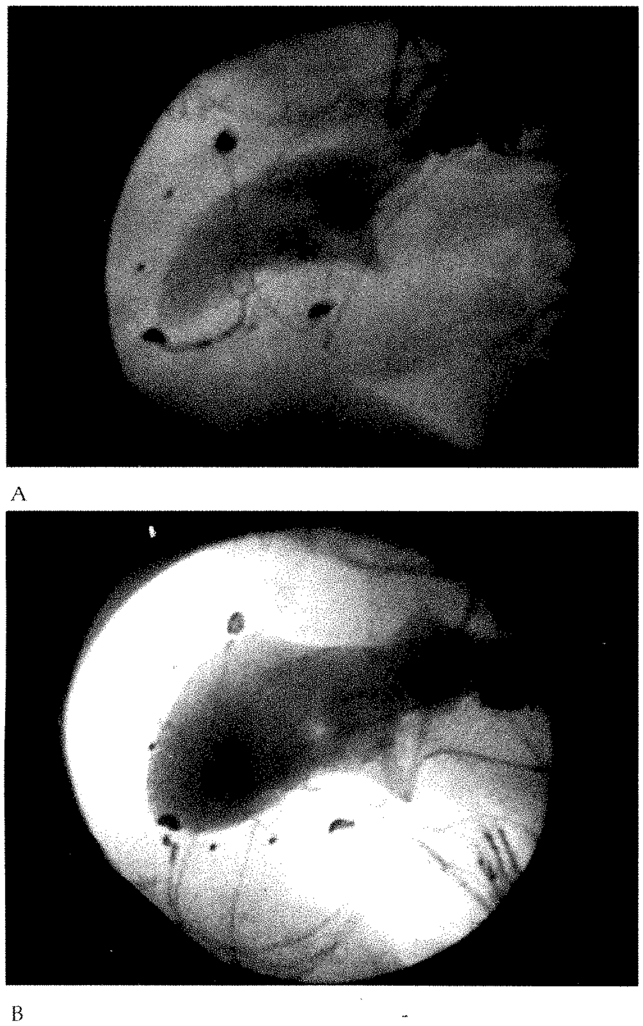

Fig 7. Right posterior oblique left ventriculograms during systole in 1 sheep before (A) and 21 days after (3) ligation of the distal ovine homonymous and second diagonal coronary arteries. Epicardial markers used for measurements summarized in Table 1 are visible. Note the expansion of the left ventricular cavity and expansion of the anteroapical ventricular wall.

preparations. Fink and Merrick [23] found a few microscopic intercoronary anastomoses in the upper ventricular and lower atrial septa of sheep. Euler and co-workers [22] observed that ligation of the circumflex coronary artery produced a sharp boundary between perfused and nonperfused tissue. After ligation of the circumflex coronary artery, dye injection of the left main coronary artery did not cause retrograde filling of the distal circumflex arterial system. Our studies also fail to demonstrate macroscopic intercoronary arterial collateral vessels. As observed in 1 sheep by Schaper [10], lack of preformed and induced collateral vessels may be the reason that ventricular aneurysms develop in sheep after ligation of apical coronary arteries.

Ventriculogram studies indicate that thinning and expansion of the area of infarction begins within four hours of coronary artery occlusion. Our observations support those of Erlebacher and associates [27], who found rapid

Table 1. Serial Measurements of Right Anterior Oblique Ventriculograms[a]

Time	LA	USA	MSA	LSA	WT1	WT2	WT3	WT4	WT5	WT6	WT7
Control (n = 7)	143.5 ± 6.6	44.2 ± 2.3	36.4 ± 3.1	19.9 ± 3.7	17.3 ± 2.4	15.8 ± 1.8	15.1 ± 1.5	10.6 ± 0.9	12.5 ± 1.2	14.7 ± 1.1	20.9 ± 2.0
Four hours (n = 7)	149.5 ± 5.5	52.2 ± 3.1	55.6 ± 1.2[b]	37.3 ± 1.6[b]	16.9 ± 2.2	8.1 ± 1.8[b]	7.5 ± 1.3[b]	7.3 ± 1.5	9.4 ± 1.2[b]	11.1 ± 1.1[b]	20.8 ± 3.2
Three days (n = 5)	137.4 ± 9.2	50.4 ± 2.8	54.7 ± 2.0[b]	40.5 ± 3.4[b]	13.4 ± 2.1	5.4 ± 2.1[b]	6.5 ± 1.8[b]	4.5 ± 2.0[b]	8.2 ± 1.26[b]	12.1 ± 1.2	18.7 ± 2.6
One week (n = 6)	140.1 ± 7.0	58.4 ± 2.4[b]	62.4 ± 3.2[b]	40.9 ± 3.6[b]	13.3 ± 3.6	6.4 ± 1.7[b]	6.4 ± 1.4[b]	6.0 ± 1.4[b]	7.6 ± 1.8[b]	12.4 ± 1.5	18.7 ± 3.2
Three weeks (n = 5)	138.3 ± 5.1	58.3 ± 3.5[b]	56.6 ± 2.3[b]	38.9 ± 4.1[b]	12.6 ± 2.7	5.0 ± 0.2[b]	5.1 ± 1.3[b]	5.9 ± 2.2[b]	6.2 ± 2.0[b]	11.8 ± 2.2	18.9 ± 4.1
Two months (n = 1)	124.3	64.8	67.4	48.3	6.5	3.4	. . .	. . .	1.8	5.2	14.0

[a] Corrected measurements of left ventricular cavity dimensions across the four axes illustrated in Figure 1 of right posterior oblique left ventricular angiograms of 14 sheep. Angiograms were obtained before and up to 60 days after ligation of the distal homonymous (left anterior descending coronary artery) and second diagonal branches. Corrected wall thickness measurements, WT1–7, at the seven points depicted in Figure 1 are also tabulated. Ligatures were tied just cephalad to point WT2. Postinfarction measurements at each time interval were compared with control measurements by the unpaired *t* statistic. [b] $p < 0.05$ by unpaired Student's *t* test.

LA = long axis of ventricular cavity; LSA = lower short axis; MSA = mid-short axis; USA = upper short axis of ventricular cavity.

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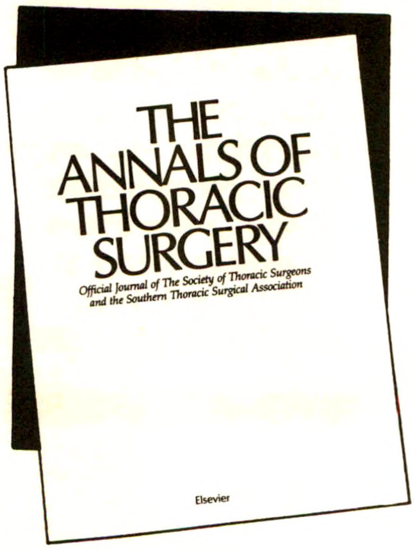
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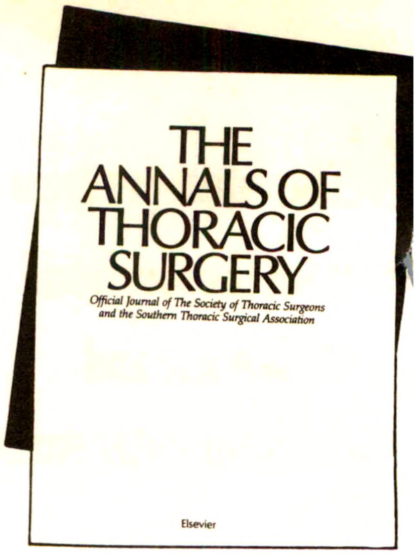
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increases in end-diastolic segment length of infarctions in canine myocardium. The reported three- to five-day delay in infarct expansion is based on postmortem studies in humans, rats, and dogs [7, 28–30]. Furthermore, the fixed epicardial reference points of our serial ventriculograms indicate that both uninfarcted muscle adjacent to infarcted myocardium and infarcted myocardium become thinner and stretched during evolution of an apical left ventricular aneurysm. In our single animal studied at 60 days, over 60% of the left ventricular contour was akinetic, as compared with the 30% akinetic segment noted at 3 weeks. This animal had clinical heart failure and a left ventricular end-diastolic pressure of 54 mm Hg.

Previous attempts to develop large animal models of left ventricular aneurysm produced inconsistent results [7, 8, 10, 30, 31] or primitive mechanical imitations [32]. Reproducible left ventricular aneurysms develop in sheep after coronary arterial ligation, and the evolution and morphological characteristics of these aneurysms are similar to clinical and postmortem observations of left ventricular aneurysms in humans [3, 6, 28, 29]. Furthermore, the ovine coronary arterial and venous anatomy simplifies studies of left ventricular metabolism. Simultaneous quantitative measurements of both left ventricular metabolites and mechanics after acute myocardial infarction may help to elucidate the pathogenesis of the process and lead to improved medical and surgical therapy.

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Tracheobronchial Rupture due to Blunt Chest Trauma: A Follow-up Study

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From 1970 to 1988, 9 patients were treated for tracheobronchial rupture arising from nonpenetrating thoracic trauma. All patients had dyspnea and pneumothorax. Four patients had rupture of the right main bronchus, 3 had rupture of the left main bronchus, 1 had rupture of the right intermediate bronchus, and 1 had rupture of the trachea. Four patients were operated on within 24 hours. Three of them had a massive air leak into the suction drainage and underwent thoracotomy. The fourth patient presented difficulties with endotracheal intubation and required a collar incision. Primary reconstruction was performed in all 4 patients. Five patients had a delay of nine to 89 days before operation. All of them had good primary healing but later developed dyspnea. Bronchoscopy revealed scar obstruction in all 5. The stenosed segment was resected in 4 patients at thoracotomy. The fifth patient, who had an intermediate bronchus rupture, underwent lobectomy. Seven patients were followed from 6 months to 18 years. One of them, who had a nine-day delay in treatment, needed further operation 6 months after the accident because of scar obstruction. The other 6 patients were interviewed, examined, and studied with spirometry, body plethysmography, bronchoscopy, and bronchography. In these 6 patients no stricture was seen, and there was no reduced pulmonary function due to the rupture even when operation was delayed.

(Ann Thorac Surg 1989;48:846–9)

Rupture of the tracheobronchial tree is uncommon after blunt chest trauma. However, the number of these patients has increased during the last decades due to an increase in traffic accidents and improved ambulance services transporting patients with major chest trauma alive to the hospital [1, 2]. The diagnosis of tracheobronchial rupture is not often clear, and it has to be considered in the management of major chest trauma. In this study our purpose is to elucidate diagnostic and therapeutic problems and to describe the long-term results of operative treatment of tracheobronchial rupture.

Material and Methods

Nine male patients aged between 15 and 50 years (mean age, 33 years), were treated for tracheobronchial rupture due to blunt thoracic trauma during the period 1970 to 1988 at the Department of Thoracic and Cardiovascular Surgery, Helsinki University Central Hospital. In six instances, the cause was a high-energy traffic accident; 3 other patients had sustained compression trauma. Two of them were pressed between a motor vehicle and a wall, and 1 had been stepped on by a horse.

On admission to the hospital all 9 patients had dyspnea; 8 had subcutaneous emphysema and 2 had flail chest. On chest roentgenogram all 9 patients had pneumothorax (in 1, it was not detected until two days after the accident), 6 patients had pneumomediastinum, 4 had hemothorax, and 4 had rib fractures. In 1 patient a pneumothorax on the right side developed two days after the accident although the rupture was in the left main bronchus (Fig 1). Another patient had a pneumoperitoneum, but no intraabdominal damage was discovered at laparotomy.

Every patient had some concomitant injuries (mean, two concomitant injuries per patient). Six patients had other life-threatening injuries: pulmonary contusion, splenic or pancreatic rupture, or luxation of the heart because of rupture of the pericardium. Minor concomitant injuries were rib, clavicle, and radius fractures.

Results

Treatment

Four patients underwent operation within 24 hours after the accident. Location of the rupture was in the right main bronchus in 4, in the left main bronchus in 3, in the trachea in 1, and in the right intermediate bronchus in 1. Indications for emergency treatment were massive air leak via the suction drainage in patients with bronchial rupture or difficulties with endotracheal intubation in the patient who had tracheal rupture. The latter patient was treated via a collar incision. The other 3 patients underwent right thoracotomy as the pneumothorax was on that side. However, 1 of these 3 had a rupture of the left main bronchus. The other 2 patients sustained a rupture of the right main bronchus. In all of these 4 patients the rupture site was repaired without any resection using absorbable material (Dexon [Davis & Geck, Gosport, Great Britain] or Vicryl [Ethicon, Sollentuna, Sweden]). Interrupted or

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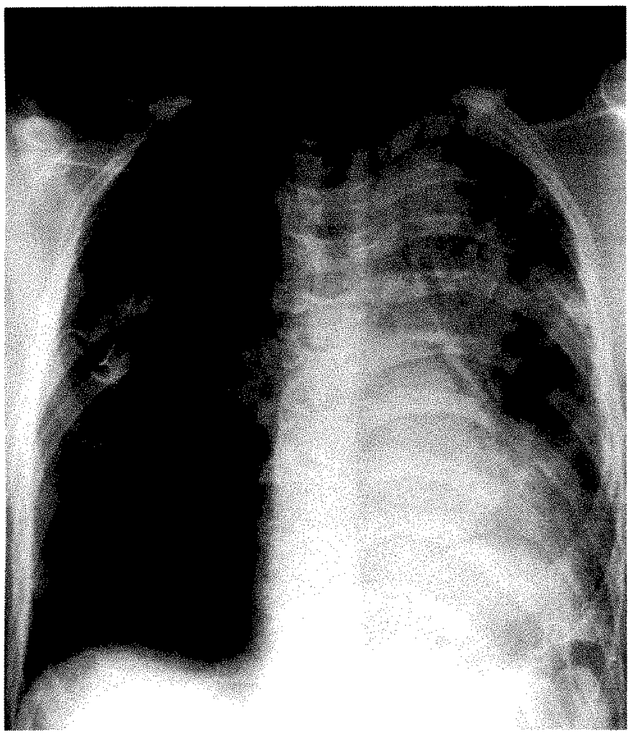

Fig 1. Right pneumothorax two days after rupture of left main bronchus.

continuous suturing was used. Suction drainage was continued from four to 17 days after bronchial reconstruction. Respirator treatment was required from one to 19 days (mean duration, seven days). The hospitalization time was from 13 to 28 days (mean, 18 days).

In 5 patients the operation was delayed nine to 89 days because of delayed diagnosis. In 4 patients the reason was complete reexpansion of the lung after two to eight days of suction drainage. In the fifth patient, who had nine days' delay, a pneumothorax did not develop until two days after the accident. However, in all 5 patients dyspnea later redeveloped, and bronchoscopy revealed obstruction and granulation tissue as a mark of rupture in the involved bronchus. Two patients had rupture of the right main bronchus, 1 had rupture of the right intermediate bronchus, and 2 had rupture of the left main bronchus. Those patients with right-sided rupture underwent right thoracotomy, and the others, left thoracotomy. In all 5 patients operation confirmed that there was marked stenosis at the rupture site. In those 4 patients in whom the rupture was in the main bronchus the stenosed segment was excised, mucous exudate was suctioned from the distal bronchus, and end-to-end anastomosis was performed. The anastomosis was performed with absorbable material in 3 patients; in the fourth patient the anastomosis was performed with nonabsorbable continuous sutures (Prolene; Ethicon, Sollentuna, Sweden). The patient with the right intermediate bronchus rupture underwent lobectomy because of infection and atelectasis. For all patients, in the phase of definitive treatment suction drainage was used for two to 13 days (mean, six days).

The patient with a tracheal rupture had recurrent nerve paralysis as a complication. There was no mortality in this series.

Follow-up

Seven patients were followed for 6 months to 18 years after the accident. The last of them, who had nine days' delay in treatment, experienced chest pain 6 months after the operation. Bronchoscopy showed total obstruction in the previously operated bronchus, and a new thoracotomy had to be done to resect the stenosed segment. The other 6 patients were called to examination, which included interview, physical examination, spirometry, body plethysmography, bronchoscopy, and bronchography. All 6 patients were smokers. None had pneumonia or bronchitis.

Bronchoscopy revealed a slight local narrowing in 1 patient, and a second patient had slight cartilage deformation at the rupture site. Two patients had mucosal redness. In no patient was granulation tissue demonstrated. Cartilage deformation was also confirmed at bronchoscopy (Table 1).

The patient who had had a ruptured trachea had some smooth deformation of the trachea according to tracheography, but it was not confirmed at bronchoscopy. The patient who had lost the right middle and lower lobes had some diffuse parenchymal infiltration in the upper lobe rendering bronchography difficult, but there were no other pathological findings at bronchoscopy.

The long-term results of the lung function studies are indicated in Table 1. Results of spirometry and body plethysmography were normal in 4 patients. One patient had moderate and 1 marked obstruction. The latter had undergone lobectomy and also had some pulmonary restriction. The patient with moderate obstruction had had no delay in the treatment of his tracheal rupture.

Comment

The incidence of tracheobronchial rupture in persons dying in accidents has been reported to be 0.85% to 2.8% [4, 5]. In traffic accidents causing blunt chest injury the incidence of tranchobronchial rupture has been 0.7% [1]. Among our patients six ruptures of nine were due to high-energy traffic accidents. Early mortality is estimated to be about 30%, but a great deal of mortality is due to multiple associated injuries [6, 7]. The mechanism of tracheobronchial rupture is probably the force acting mostly at the tracheobronchial bifurcation when the thorax is compressed [7] or the sudden increase in intrabronchial pressure against a closed glottis [8]. These theories are in concordance with the finding that the rupture is most often situated in the main bronchi [5–9] within 2.5 cm of the carina. We had similar findings in 7 patients.

Clinical manifestations are nonspecific and usually include dyspnea and subcutaneous emphysema. The chest roentgenogram often reveals mediastinal emphysema and massive pneumothorax with failure of reexpansion of the lung. Very pathognomonic for main bronchus rupture is a drop of the superior margin of the collapsed lung to below the level of the transected bronchus [2, 10–14]. However,

Table 1. Clinical Data and Lung Function Follow-up Findings After Blunt Tracheobronchial Rupture

	Patient No.						
Variable	1	2[a]	3	4	5	6	7
Age (yr)	26	35	16	50	42	50	15
Delay of operation (days)	89	40	30	0	1	0	9
Follow-up time (yr)	18	16	8	8	3	2	0.5
Bronchoscopic findings	Cartilage deformation	Mucosal redness	Normal	Mucosal redness	Normal	Local narrowing	Stenosis
Bronchography	Normal	Diffuse infiltration in right lung	Normal	Local undulation	Normal	Normal	. . .
Pulmonary function							
VC (L)	7.07	3.01	5.42	4.90	7.56	3.62	. . .
VC (%)[b]	126	60	96	108	117	175	. . .
FEV_1 (L)	5.05	1.71	5.00	2.90	6.37	3.48	. . .
$FEV_1/FVC \times 100$	72	68	95	71	87	86	. . .
RV (L)	3.22	1.79	1.20	2.88	1.73	2.13	. . .
RV (%)[b]	136	85	64	133	65	101	. . .
R_{aw} ($kPa \cdot s \cdot L^{-1}$)	0.73	0.35	0.11	0.28	0.08	0.075	. . .
R_{aw} (%)[b]	64	256	89	216	72	58	. . .
PEF (L/s)	10.76	3.43	11.18	7.20	13.96	8.64	. . .
PEF (%)	100	34	102	75	124	87	. . .
Obstruction	. . .	Marked (restriction in addition)	. . .	Moderate	. . .	. . .	. . .

[a] This patient underwent lobectomy; [b] Predicted values according to Viljanen and associates [3].

FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity; PEF = peak expiratory flow; R_{aw} = resistance in airways; RV = residual volume; VC = vital capacity.

none of our patients had this sign. A very peculiar phenomenon of left main bronchus rupture was that 2 of 3 patients had massive air leakages to the right side and only 1 to the left. The tissue surrounding the left main bronchus is so tight that it can prevent leakage to the left.

In emergency cases there is no time for bronchoscopy or other time-consuming investigations for dyspnea. When bronchoscopy was done it revealed the diagnosis in all 5 patients. Therefore, routine use of bronchoscopy, if possible, is advocated for every patient suspected of having a tracheal or bronchial rupture.

Because of lack of specific findings the diagnosis is delayed in 25% to 68% of patients [9, 11, 14, 15]. Among our patients the diagnosis was overlooked in the initial phase in 55%. One reason for delay is that peribronchial tissue may remain sufficiently intact to permit ventilation in the involved lung. However, bronchial obstruction can develop in 2 to 6 weeks due to granulation tissue at the site of rupture. Distal to the obstruction, atelectasis and infection lead to dyspnea [6, 14, 15]. Most of the reported repairs of a ruptured bronchus have been done with nonabsorbable sutures with good success [2, 10, 11, 14]. However, we have mostly used absorbable materials because of the danger of suture granulomas [16]. Scar obstruction developed in the only patient in whom we used nonabsorbable material (Prolene).

When the diagnosis is delayed and stenosis develops the stenotic segment can be excised and the bronchus reconstructed after exudate is suctioned from the distal bronchus. However, if bronchial injury is extensive or infection severe, or the rupture is situated in a small bronchus, as in 1 of our patients, resection may be necessary [2, 7, 11, 17]. Normal pulmonary function cannot be expected after a delay of 6 months, probably because of fibrosis [18]. On the other hand, successful results have been reported after 9 and 11 years delay [19, 20]. In our series we did not find reduced pulmonary function due to rupture, if the patient who lost his middle and lower lobes is excluded. We could not demonstrate anatomical or functional differences in the patients treated immediately or in those treated after a delay. Although bronchial rupture can be treated successfully in the acute or the delayed phase, early diagnosis and treatment minimizes the risk of infection and resection and saves the patient from extra hospital days.

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Percutaneous Cricothyroidostomy (Minitracheostomy) for Bronchial Toilet: Results of Therapeutic and Prophylactic Use

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In the period 1984 to 1988, 2,448 patients underwent major thoracic operations in our unit. Of these patients, 144 (5.9%) underwent percutaneous cricothyroidostomy (minitracheostomy) using a 20F pediatric silver tracheostomy tube. Minitracheostomies were performed for the treatment of sputum retention in 81 patients, prophylactically in 62 patients, and as a route for high-frequency jet ventilation in 1 patient. Minitracheostomy tubes remained in situ a median of four days. Suction function was satisfactory in 99% of patients, with only 2 patients requiring subsequent suction bronchoscopy and no occurrence of tube blockage. Ninety-four percent of minitracheostomies performed were entirely uneventful. Bleeding was the most common complication (3.5%), and no instances of subglottic stenosis have occurred. During the review period, there was a significant increase in the percentage of patients requiring major operations undergoing prophylactic, but not therapeutic, minitracheostomy ($p < 0.001$). This was associated with a significant decrease in the percentage of patients requiring suction bronchoscopy ($p < 0.001$). We conclude that prophylactic minitracheostomy with the 20F pediatric silver tracheostomy tube is a safe and effective procedure in the prevention of postoperative sputum retention.

(Ann Thorac Surg 1989;48:850–2)

Sputum retention is a common problem after thoracic operations and, if neglected, may lead to respiratory failure, bronchopneumonia, and death. The development of percutaneous cricothyroid intubation by Matthews and Hopkinson [1] has revolutionized the management of this condition, and a commercial kit is available that employs a modified 4-mm noncuffed Portex pediatric endotracheal tube as the conduit for endotracheal suction. However, the small internal diameter of the Portex tube (4 mm) only allows the passage of a 10F suction catheter, which we have found to slide poorly along the plastic tube. Difficulty in clearing viscid bronchial secretions and tube blockage may result, limiting the effectiveness of the kit as a means of improving bronchial toilet. Our experience of these difficulties prompted us to employ a 20F pediatric silver tracheostomy tube for percutaneous cricothyroidostomy (minitracheostomy). We report here our experience of 144 such procedures performed over a 5-year period.

Patients and Methods

Apparatus

The pediatric silver tracheostomy tube consists of three components: the tracheostomy tube proper, with built-in flanges for fixation tapes, a removable inner tube that facilitates tube cleaning and prevents tube blockage, and an obturator that allows easy insertion.

Technique

Minitracheostomy may be performed under local or general anesthesia, in the ward or in the operating room. All the equipment is reusable, apart from a disposable blade. The technique is essentially the same as that developed by Matthews and Hopkinson [1], except that an introducer is not necessary due to the rigidity of the silver tube. A midline vertical stab incision is made in the skin overlying the cricothyroid membrane with a No. 11 blade, and deepened through the membrane. Care is taken to avoid any subcutaneous veins. A distinct give is felt as the blade penetrates the cricothyroid membrane, after which there should be no further resistance to the passage of the blade. A pair of Dunhill artery forceps is then introduced into the larynx through the tract so created, and dilation of the tract is carried out in the vertical and horizontal planes. The pediatric silver tracheostomy tube can now be introduced into the trachea, usually without much resistance if the tract has been adequately prepared. Once the tube is in place the bronchial tree is suctioned with a 14F catheter and the tube is secured with tapes passed around the metal flanges. After suction, the inner tube is placed inside the tracheostomy tube, and the lumen is occluded with a spigot. Alternatively, the lumen may be left open and used for delivery of humidified oxygen to the patient via a standard tracheostomy mask.

The indications for prophylactic minitracheostomy that we have adopted are: poor pulmonary function in patients undergoing major thoracic operations, major lung resection associated with recurrent or phrenic nerve injury, major thoracic operation in the elderly, or a combination of these. Contraindications are an impalpable

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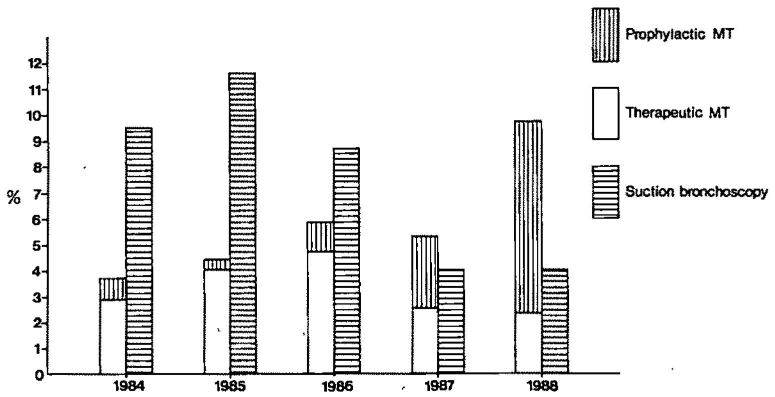

Fig 1. Changing trends in the use of prophylactic and therapeutic minitracheostomy (MT) *and suction bronchoscopy, 1984 to 1988 (percentage of patients requiring major operations in whom procedure is performed).*

cricothyroid membrane in a short, fat neck, or a hemorrhagic diathesis.

Patients

In the years 1984 to 1988, 2,448 patients underwent major thoracic operations in our unit. Of these patients, 144 (101 male, 43 female) had minitracheostomy performed for bronchial toilet. Their mean age was 66.4 years (range, 43 to 87 years); mean forced expiratory volume in 1 second was 1.76 L (range, 0.4 to 3.55 L); mean forced vital capacity was 2.96 L (range, 1 to 5.16 L); and mean ratio of forced expiratory volume in 1 second to forced vital capacity was 0.60 (range, 0.32 to 0.82). Minitracheostomy tubes remained in situ for a mean of 5.8 days (median, 4 days; range, 1 to 42 days). Reintubation was required for 2 patients after removal, both for recurrent sputum retention.

The majority of patients (110 patients, 76%) had lung resection for malignant disease. The remainder had esophageal resection for carcinoma (14), lung resection for benign disease (10), and miscellaneous thoracic operations (10), including a myasthenic patient who underwent thymectomy.

Sixty-two minitracheostomies (43%) were performed prophylactically in the operating room at the end of the operation and simultaneously with extubation. Eighty-one minitracheostomies (56%) were performed for established sputum retention; initially this tended to be as an adjunct to suction bronchoscopy, but later minitracheostomy was used on its own as definitive treatment. In 1 patient with a bronchopleural fistula after pneumonectomy, minitracheostomy was performed for high-frequency jet ventilation. Of the 144 patients who underwent minitracheostomy, only 2 required subsequent suction bronchoscopy.

Results

Figure 1 illustrates the changing trends of prophylactic and therapeutic minitracheostomy and suction bronchoscopy in the period under study. The percentage of total patients requiring major operations undergoing minitracheostomy increased significantly from 1984 to 1988 ($p <$ 0.001, standard error of difference between percentages), while the percentage undergoing suction bronchoscopy decreased significantly ($p < 0.001$). Furthermore, although the percentage of therapeutic minitracheostomies performed did not change significantly from year to year, that of prophylactic minitracheostomies increased significantly from 1984 to 1988 ($p < 0.001$).

Table 1 illustrates complications associated with minitracheostomy. In the great majority of patients (94%), minitracheostomy was an entirely uneventful procedure. Bleeding occurred in 5 patients: 1 stopped bleeding spontaneously, 2 stopped after the minitracheostomy was removed, 1 required revision to formal tracheostomy, and 1 sustained airway obstruction by clots with consequent hypoxic cardiac arrest from which he was successfully resuscitated. Most stomas were sealed within three to five days of tube removal; however, in 3 patients prolonged air leak occurred (up to 13 days in 1). All stomas were completely healed when reviewed at our routine 6-week follow-up clinic. Subglottic stenosis was not detected clinically in any patient.

Comment

The Portex "Mini-Trach" system as devised by Matthews and Hopkinson [1] has proved deservedly popular in the prophylaxis and treatment of sputum retention. Early problems with tube dislocation [2] have been countered by the addition of a flange. Our own experience with this device has revealed further problems with inadequate suction function and tube blockage, both of which were reported by Matthews and Hopkinson in their original paper [1]. The 10F suction catheter did not prove adequate for the viscidity and quantity of bronchial secretions in our patient population; furthermore, undue friction between the plastic of the suction catheter and the Portex tube created extra difficulties. The Portex system also did not provide for tube cleaning, leading to inspissation of

Table 1. Complications of Minitracheostomy

Complication	No. of Patients
Early	
None	136 (94.4%)
Hemorrhage	5 (3.5%)
Prolonged air leak	3 (2.1%)
Late	
Stomal skin tethering	1

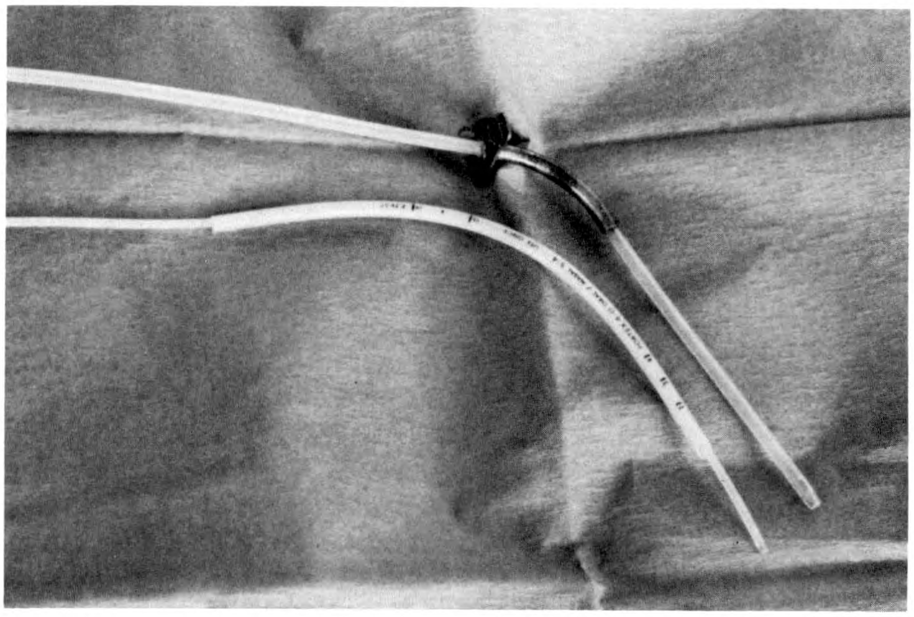

Fig 2. Demonstration of the much larger suction catheter that can be passed down the 20F pediatric silver tracheostomy tube, despite similar external diameter to the Portex "Mini-Trach" tube.

secretions in the lumen of the tube and subsequent blockage. These problems prompted us to employ the 20F pediatric silver tracheostomy tube. Although it possessed much the same external diameter as the Portex tube (6.7 mm versus 5.4 mm), it allowed easy passage of a 14F suction catheter (Fig 2). Furthermore, tube blockage was prevented by the presence of an inner tube that could be removed for cleaning. Suction function was greatly enhanced, and the system had the extra advantage of being reusable.

The efficacy of our technique is shown by the observation that the increase in use of minitracheostomy has been associated with a significant reduction in the incidence of postoperative suction bronchoscopy. This may be the result of the effectiveness of minitracheostomy in the prophylaxis of postoperative sputum retention or of the use of minitracheostomy instead of suction bronchoscopy in the treatment of sputum retention, or both. Consideration of our results, however, shows that the former is the likelier explanation, for while the incidence of minitracheostomy performed for sputum retention has not changed significantly over the years, the incidence of prophylactic minitracheostomy has increased ($p < 0.001$).

The low incidence of complications in our series compares favorably with that of formal tracheostomy, for which morbidity rates of up to 40% [3, 4] are quoted. In particular, serious operative hemorrhage occurs in up to 5% of tracheostomies [5], with delayed massive hemorrhage from great vessel necrosis in 1% [6]. By contrast, formal cricothyroidostomy has a low morbidity similar to minitracheostomy [7], although by its very nature the procedure is more invasive. Earlier concern about the potentially serious long-term complication of subglottic stenosis after formal cricothyroidostomy has largely been dispelled by more recent studies [7, 8], and it is our observation as well as that of others [9, 10] that such a complication does not occur with the insertion of small-bore minitracheostomy tubes in adults.

The one serious complication in our series was the development of clot obstruction of the airways in a patient who should, in retrospect, have had his tube removed as the lack of a cuff leaves the airway unprotected from blood. Since that incident it has been our policy to remove the tube and proceed to neck exploration/tracheostomy for persistent bleeding, and we have had no recurrence of this complication.

We conclude that percutaneous cricothyroidostomy with the 20F pediatric silver tracheostomy tube is a safe and effective procedure, and is of value in the prevention of postoperative sputum retention.

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Combined Coronary Revascularization and Splenectomy

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Idiopathic thrombocytopenic purpura is rarely associated with coronary artery disease. In this report, we describe the successful surgical management of a patient with idiopathic thrombocytopenic purpura and angina pectoris.

(Ann Thorac Surg 1989;48:853–4)

Idiopathic thrombocytopenic purpura is characterized by a decreased number of platelets and excessive posttraumatic bleeding. Rarely is it associated with coronary artery disease. We report the successful surgical management of a patient with idiopathic thrombocytopenic purpura and severe coronary artery disease by splenectomy and coronary artery bypass grafting (CABG).

A 37-year-old man was admitted to Kitano Hospital in July 1988 because of severe chest pain and petechiae. Laboratory data included a platelet count of 8,000/mL, a bleeding time of 25 minutes, and normal coagulation factors. The bone marrow contained an increased number of megakaryocytes. Coronary angiography showed 75% stenosis at the proximal portion of the left anterior descending coronary artery, total obstruction of the obtuse marginal branch, and 99% stenosis of the right coronary artery.

The patient was transferred to Osaka Medical College for CABG. A midline skin incision was extended to just above the umbilicus, and the left internal mammary artery was taken down. Through a median laparotomy, the spleen was removed without difficulty, and then the right gastroepiploic artery was freed along the greater curvature of the stomach. Because the posterior wall of the left ventricle was already infarcted, we decided to use a bovine internal mammary artery biograft (Bioflow) instead of a saphenous vein graft to avoid a leg wound. Triple CABG (left internal mammary artery–left anterior descending coronary artery, right gastroepiploic artery–right coronary artery, Bioflow–obtuse marginal branch) was done using standard cardiopulmonary bypass with 53 minutes of aortic cross-clamp time (Fig 1).

Just after the termination of cardiopulmonary bypass, packed platelets were transfused, and hemostasis was carefully achieved. The platelet count increased to 242,000/mL on the next day, and the thoracic drainage tubes were removed on the fifth postoperative day. During hospitalization, the platelet count remained around 300,000/mL with steroid therapy (Fig 2). The postoperative course was uneventful. When discharged, the patient was free from both angina pectoris and the tendency to bleed.

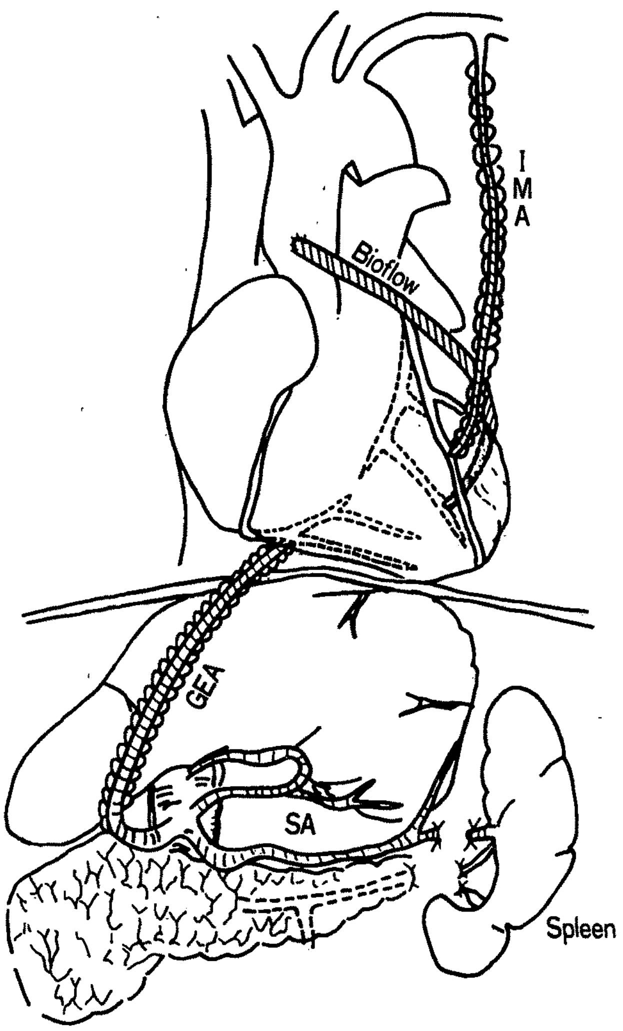

Fig 1. Combined triple coronary artery bypass grafting and splenectomy. (GEA = *gastroepiploic artery;* IMA = *internal mammary artery;* SA = *splenic artery.)*

Comment

A survey of the literature revealed that a few patients with ischemic heart disease and idiopathic thrombocytopenic

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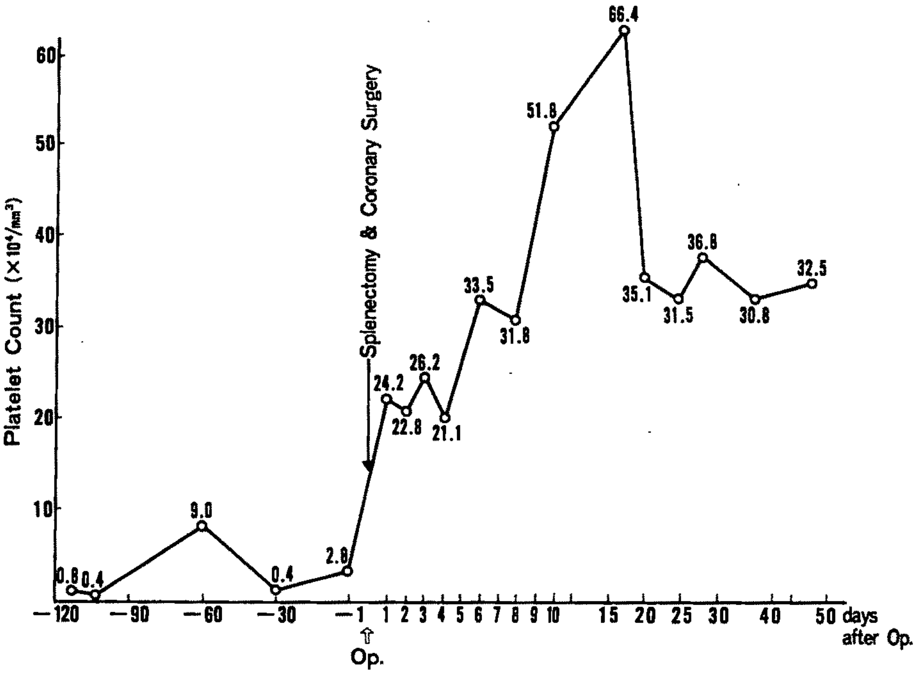

Fig 2. Change in platelet count. (Op. = operation.)

purpura have undergone CABG without concomitant splenectomy [1]. We are aware of only one report of combined splenectomy and mitral valve replacement in a patient with idiopathic thrombocytopenic purpura and mitral regurgitation [2]. To our knowledge, the present report is the first of combined splenectomy and CABG.

Medical treatment such as corticosteroids or γ-globulin results in a low percentage of patients with sustained clinical remission of idiopathic thrombocytopenic purpura [3]. As an ideal surgical procedure, we performed a one-stage operation involving splenectomy and CABG. The platelet count normalized within a day, and no bleeding tendency was found postoperatively.

Because of the young age of the patient and the possible long-term administration of steroids, we used the left internal mammary artery and the right gastroepiploic artery for the area of viable myocardium [4, 5]. The right gastroepiploic artery is a particularly suitable conduit in patients requiring a laparotomy for splenectomy, for example, as illustrated in the case of our patient. Its dissection is safe and easily accomplished without serious invasion. Also, the right gastroepiploic artery can be separated readily without bleeding and is long enough to reach the right coronary artery.

We thank Dr Mamoru Hiyama, Hiratsuka Gastrointestinal Hospital, for helping with the splenectomy and Dr Takashi Umezawa, Dr Takao Ishimura, and Dr Akira Wakabayashi, Department of Cardiology, Kitano Hospital, for referring this patient to us.

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Ultrasonic Debridement of Calcified Pericardium in Constrictive Pericarditis

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Debridement of heavily calcified and adherent pericardium with an ultrasonic surgical aspiration device is described. The device proved to be a useful adjunct to the complete debridement of densely adherent pericardium.

(Ann Thorac Surg 1989;48:855–6)

Extensive calcification of the pericardium is a well-known complication of pericarditis. The development of constrictive pericardial physiology may necessitate pericardiectomy. Complete resection of the pericardium is desirable [1], but can be compromised by the presence of densely adherent, calcified pericardium. In an attempt to separate the parietal pericardium from the visceral surface of the heart, it may be necessary to leave substantial amounts of calcified plaques over the epicardium to avoid risk of injury to the myocardium or vessels. In view of these technical difficulties, we elected to employ a hand-held ultrasonic surgical aspirating device (Cavitron ultrasonic surgical aspirator [CUSA] System 200 console; Cavitron Surgical Systems, Inc, Stamford, CT) to debride the pericardium in a patient with calcific constrictive pericardial disease.

A 43-year-old man was seen with complaints of "neck fullness" during exercise 1 year before admission. A chest roentgenogram at that time revealed substantial pericardial calcification. Over the next year, the symptoms increased. The patient began to limit his activities and was reevaluated. He gave no history of an illness suggestive of acute pericarditis, rheumatic illness, or prior fungous diseases. He was transferred to our institution with the diagnosis of constrictive pericardial disease.

On examination, he had a resting brachial blood pressure of 112/78 mm Hg, and was found to have major jugular vein distention to the mandible at 30 degrees' elevation. Hepatic pulsations were palpable after moderate exercise (12 sit-ups). There was only a trace of peripheral edema. The serum blood urea nitrogen to creatinine ratio was 22/1.3.

Catheterization was performed. Hemodynamic study revealed equalization of the left and right ventricular diastolic pressures at approximately 18 mm Hg during simultaneous recording. Prominent x and y descents were noted in the right atrial waveform, and a characteristic "square-root" appearance of the right ventricular waveform was noted. Plain chest roentgenograms showed extensive calcification of the pericardium (Fig 1). The diagnosis of chronic constrictive pericardial disease was made, and complete pericardiectomy was planned. Informed consent, including discussion of the probable use of the Cavitron ultrasonic surgical aspirator was obtained.

The operation was performed through a median sternotomy. The pericardium was heavily calcified and quite adherent to most of the heart. Calcific involvement was particularly severe at the inferior cavoatrial junction, over the anterior right ventricle, and over the diaphragmatic surface of the heart. After incision of the pericardial sac, the ultrasonic device was brought into use.

The Cavitron ultrasonic surgical aspirator is an electrically powered, hand-held device with a tip that vibrates at a frequency of approximately 23,000 times per second.

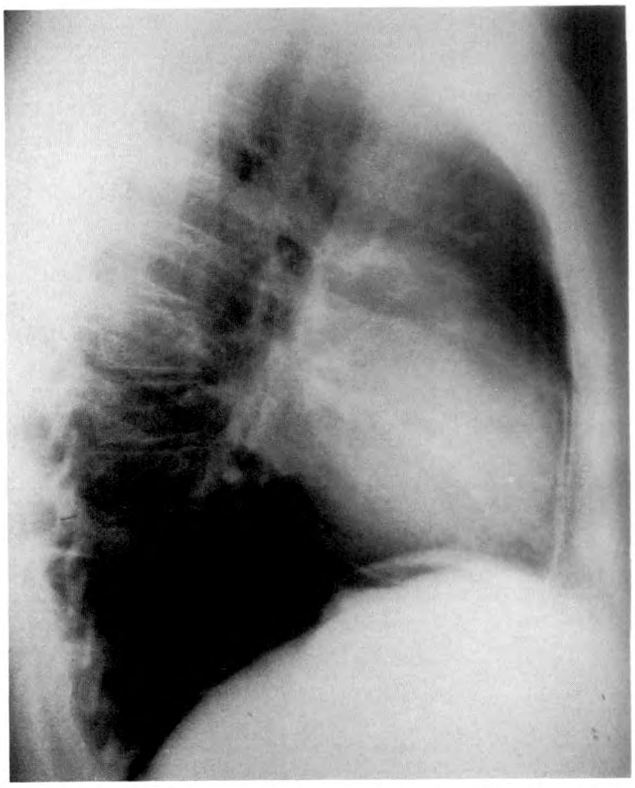

Fig 1. Preoperative lateral chest roentgenogram showing inferior pericardial calcification.

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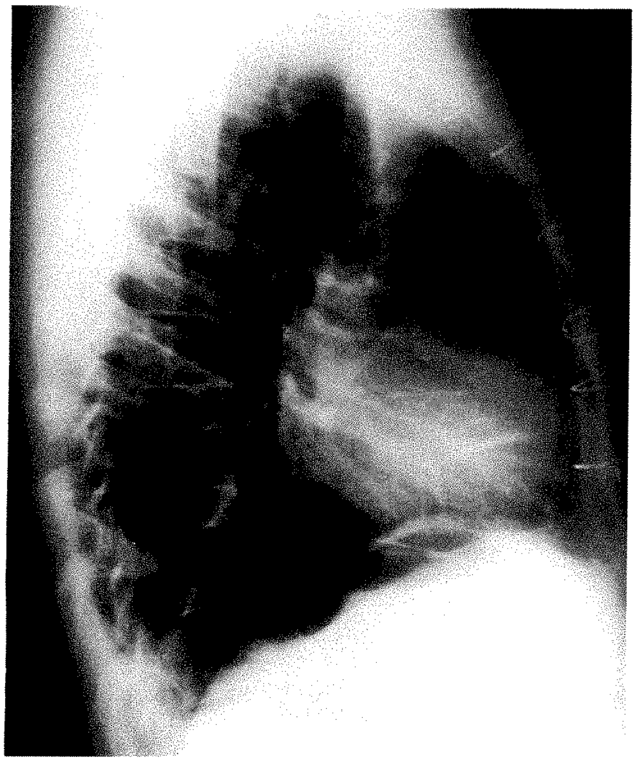

Fig 2. Postoperative lateral chest roentgenogram showing removal of virtually all calcium.

Irrigation and suction are provided at the tip. Intermittent irrigation and use of the ultrasonic dissecting tip can be controlled by the operator. Both the amount of suction and the amplitude of tip variation are adjusted on the console apart from the operative field. It was used at approximately 80% to 90% of its maximum amplitude, and 50% of its maximum suction.

We used the ultrasonic device to make a plane between the surface of the heart and the calcified pericardium. Most of the calcified material could be elevated from the epicardium or myocardium and removed in sheets. Some of the calcium, however, was so adherent to the myocardium as to preclude resection of calcium in layers. This material could be pulverized by the ultrasonic device without producing any myocardial or epicardial bleeding. Several coronary branches were stripped of their epicardial fat and all surrounding calcium without bleeding. The area around the inferior cavoatrial junction was particularly easy to debride with this device. Irrigation and suction were provided primarily from conventional sources, rather than from the device itself.

Cardiopulmonary bypass was not required. The patient tolerated the procedure very well with minimal blood loss and no blood replacement. A pulmonary artery catheter did demonstrate a slight decrease in central venous pressure and pulmonary diastolic pressure. Twelve hours later, immediately before catheter removal, right heart filling pressure was 13 mm Hg and pulmonary diastolic pressure was 12 mm Hg. Histological evaluation of the pericardium revealed only chronic inflammation. A chest roentgenogram obtained on the fifth postoperative day before discharge showed virtually complete removal of the pericardium (Fig 2).

In the 9 months since the procedure, the patient has been able to resume vigorous physical activities without limitation. A recent evaluation revealed no jugular vein distention and no pericardial rub or cardiac murmur.

Comment

The ultrasonic device used in this patient is commonly employed by neurosurgeons for the resection of brain tumors and by general surgeons for hepatic resections. The device is noted for its ability to resect soft tissue without harming blood vessels. It has not yet been approved by the Food and Drug Administration for marketing as a surgical tool in the field of cardiac surgery. Its use has been reported for the division of a myocardial muscle bridge [2]. It has also been used by us and others (Bruce P. Mindich, MD, New York, NY: personal communication, 1989) for the successful debridement of calcified aortic valve leaflets. These new applications stimulated our interest in testing the device in this patient with calcific pericardial constriction. The device was equally effective at removing fibrotic and calcified material.

For most cases of constrictive pericarditis, standard techniques of dissection are safe and effective. Occasionally, however, dense adherence of the pericardium to the underlying epicardium, and even infiltration of the myocardium by calcium, present the surgeon with the choice of safe, though inadequate dissection versus a more complete pericardiectomy and the hazards of myocardial and coronary artery injury [1]. This particular patient represented the far end of the spectrum with respect to the degree of calcification and dense pericardial fusion. The successful management of this patient suggests that the ultrasonic surgical aspiration device is extremely effective in performing a safe, complete pericardiectomy.

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Tricuspid Valve *Candida* Endocarditis Cured by Valve-Sparing Debridement

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This report describes the case of a patient in whom *Candida albicans* tricuspid valve endocarditis was treated by excision of a large vegetation and partial tricuspid valvectomy without prosthetic valve replacement. In select patients, valve-sparing debridement may allow a long-term cure and may be the procedure of choice in the treatment of *Candida* tricuspid valve endocarditis.

(Ann Thorac Surg 1989;48:857–8)

Fungous endocarditis has a high mortality despite aggressive surgical and medical treatment [1]. The conventional treatment of fungous endocarditis with valvular vegetations is early excision of the diseased valve followed by prolonged treatment with antifungus drugs. In the tricuspid position, the infected valve can be excised without valve replacement, although some patients will require late valve replacement [2]. We report the case of a patient in whom *Candida albicans* endocarditis of the tricuspid valve was treated by excision of a large vegetation with preservation of most of the unaffected native valve.

A 22-year-old woman underwent a subtotal colectomy for ulcerative colitis on August 1, 1985. Postoperatively she had a spiking temperature to 39°C or 40°C every day. *Candida albicans* was recovered from multiple blood cultures. Intravenous administration of amphotericin B was begun.

Despite continuation of the antifungus treatment, the patient noted dyspnea and pedal edema early in November. A chest roentgenogram showed a cardiothoracic ratio of 68% and a pleural effusion on the left side. Two-dimensional echocardiography disclosed a large vegetation attached to the anterior leaflet of the tricuspid valve and approximately 4 × 4 cm in cross section (Fig 1A). From these findings, it was easily surmised that the large vegetation was disturbing the inflow of blood through the tricuspid valve and would result in congestive heart failure.

Because of both the patient's rapidly deteriorating clinical condition despite appropriate antibiotics and the echocardiographic demonstration of the large vegetation, surgical intervention was undertaken. At operation, a large (4 × 4 × 4 cm) vegetation was found adherent to the anterior leaflet of the tricuspid valve close to the posterior

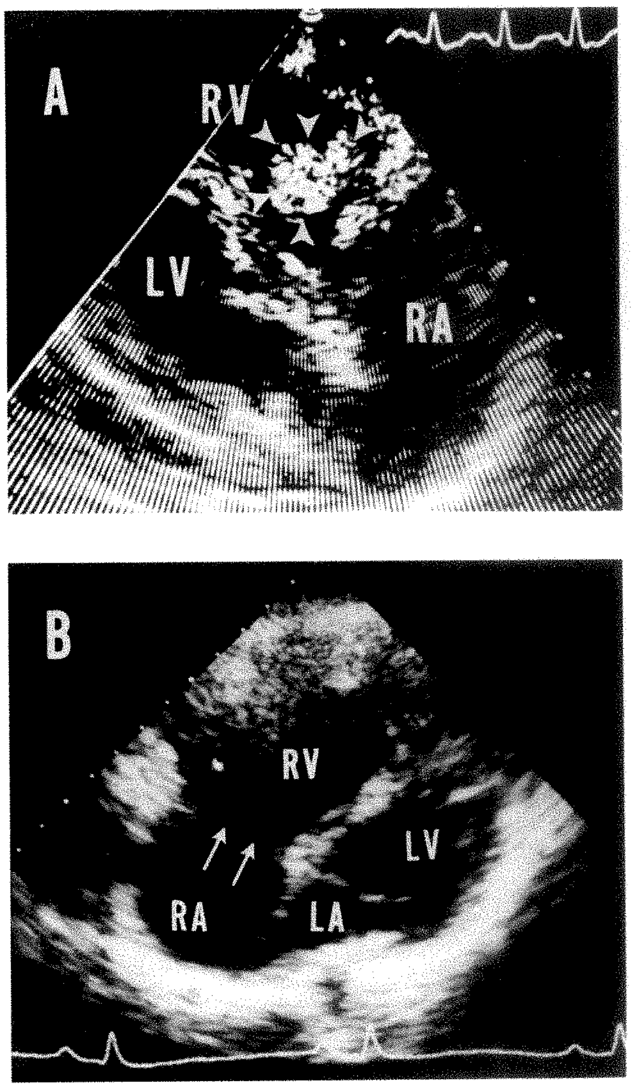

Fig 1. (A) Preoperative two-dimensional echocardiogram demonstrating a large vegetation (arrowheads) *on the tricuspid valve. (B) Two-dimensional echocardiogram 30 months after operation demonstrating the tricuspid valve* (arrows) *to be free from vegetation.* (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

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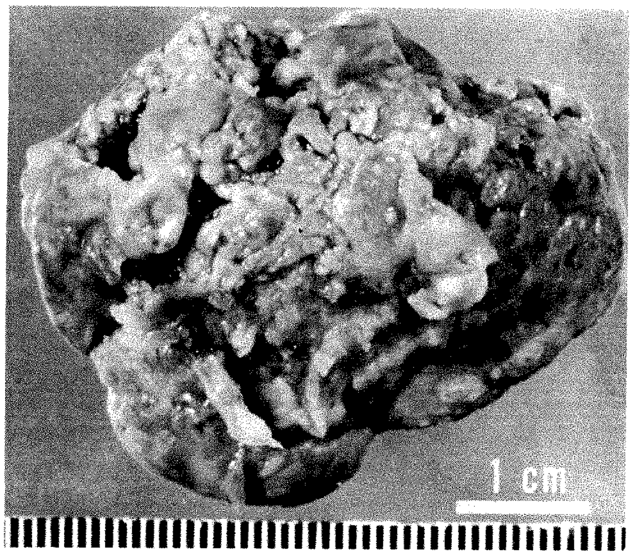

Fig 2. The surgically resected fungous vegetation.

leaflet (Fig 2). The vegetation was excised completely together with the infected half of the posterior leaflet and the infected third of the anterior leaflet. All of the vegetation was excised without valve replacement. Histopathological study confirmed that the vegetation was due to fungus, and *C albicans* was identified in the removed tissue and in cultures (Fig 3).

Postoperatively, a total cumulative dose of 1.5 g of amphotericin B was administered. In addition, the following daily regimen was begun: 600 mg of miconazole intravenously and 6 g of 5-fluorocytosine orally. All blood cultures grown in the postoperative period were sterile, and repeat two-dimensional echocardiography 30 months after operation demonstrated no recurrence of the vegetation (Fig 1B). Three years after operation, the patient is well, without any symptoms of congestive heart failure, although pulsed Doppler echocardiography shows the presence of minimal tricuspid regurgitation.

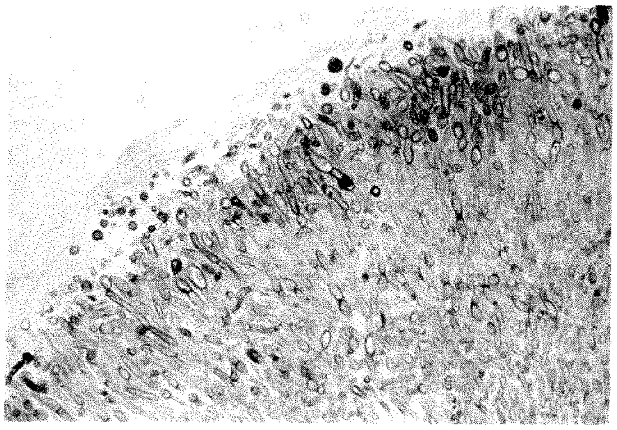

Fig 3. Histological section of the debrided vegetation showing Candida *pseudohyphae. (Periodic acid–Schiff; ×200 before 32% reduction.)*

Comment

In patients with infective endocarditis of the tricuspid valve, medical treatment carries a favorable prognosis with cure rates of up to 90% [3]. However, some patients will require operation for uncontrolled sepsis, cardiac failure, or recurrent emboli. Surgical treatment involves valvectomy with or without prosthetic valve replacement [2, 4]. In 1986, Pruett and co-workers [5] described the simple excision of a fungous vegetation from a tricuspid valve. In three other reports [6–8], nonfungous endocarditis of the tricuspid valve was successfully treated with valve-sparing debridement. The use of this method is limited to those patients in whom a well-circumscribed vegetation is associated with little or no damage to the valve. In select patients without substantial valve destruction, debridement that spares as much of the native valve as possible may have greater benefits than total valve resection, which has previously been performed.

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Solitary Plasmacytoma of the Sternum: Surgical Resection With Long-Term Follow-up

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Two patients with solitary plasmacytoma of the sternum were treated with primary surgical resection. Good long-term results were obtained. There is no long-term documentation that primary radiotherapy alone is the treatment of choice for solitary plasmacytomas. An individualized approach is warranted, especially when there is an isolated bulky lesion involving the chest wall.

(Ann Thorac Surg 1989;48:859–62)

Primary neoplasms of the chest wall represent nearly 8% of all intrinsic bone tumors [1]. Approximately 50% are malignant [2]. Primary tumors of the sternum are generally malignant [3]. Solitary plasmacytomas are rare tumors of the bony chest wall. Radiation therapy has proved to be satisfactory treatment for most solitary plasmacytomas or extramedullary plasmacytomas [4]. We present the cases of 2 patients with solitary plasmacytoma without evidence of multiple myeloma who were effectively treated with primary surgical excision and reconstruction.

Patient 1

A 53-year-old man was seen in St. Joseph Hospital with anterior chest wall pain on June 19, 1972. Pain had occurred intermittently over the same area for the previous 4 years after blunt chest trauma. A chest roentgenogram revealed a pathological sternal fracture. Tomograms showed a midsternal bony lesion with disruption of the cortices. Physical examination was normal aside from a visible nonpulsatile, tender midsternal mass. Serum protein electrophoresis was normal with no presence of Bence Jones proteins. A skeletal survey showed no evidence of malignancy or myeloma.

On June 22, 1982, en bloc resection of the lower sternum was performed. Overlying skin and subcutaneous tissue were not removed. The anterior chest wall defect was reconstructed with Marlex mesh. The upper manubrium was left along with attached clavicles. The postoperative course was uneventful. The final pathology report confirmed solitary plasmacytoma of the sternum. Uninvolved portions from the sternum and ribs had normal marrow. At 16 years, the patient has no further evidence of local or distant disease and no evidence of myeloma.

Patient 2

A 69-year-old man was seen at Fitzsimons Army Medical Center on October 2, 1985, with an anterior upper chest wall mass. Swelling of the right sternoclavicular area had been noted 5 years earlier. The mass had increased during the preceding 6 months. There was no pain or discomfort. Physical examination was normal aside from a soft, nontender, fixed mass 15 × 10 × 5 cm involving the anterior upper sternum (Fig 1A). There were no pulsations or bruits. Complete blood cell count and 12-channel chemical profile were normal. The 24-hour urine study was negative for Bence Jones proteins. Serum and urine protein electrophoresis revealed an immunoglobulin A monoclonal gammopathy. Chest roentgenograms showed an anterior soft-tissue mass extending into the right supraclavicular area. There was extensive bony destruction of the manubrium. This was confirmed by computed tomography. A percutaneous needle biopsy was done, and plasmacytoma was identified. Iliac bone marrow aspirate revealed no evidence of myeloma. A computed tomogram of the abdomen, a bone scan, and a skeletal survey of the skull and bones were negative.

Initially, primary radiotherapy was planned. The patient received 2,600 rads to the anterior chest wall without clinically significant change, and further radiotherapy as primary treatment was stopped. Therefore, on November 22, 1985, en bloc resection of skin, subcutaneous tissue, muscle, manubrium, medial bilateral clavicles, and thymus was performed. The posterior defect was repaired with Marlex mesh. Bilateral pectoralis muscle flaps were fashioned to close the defect. The operation was completed with a skin graft over the muscle flaps.

Aside from early respiratory failure, the patient made an uneventful recovery. The final pathology report confirmed solitary plasmacytoma with uninvolved margins (Fig 2). Three years after operation, the patient is alive and well with no evidence of local or distant recurrence or myeloma (Fig 1B).

Comment

Multiple myeloma is a disseminated neoplastic monoclonal gammopathy. Localized plasmacytoma is also a mono-

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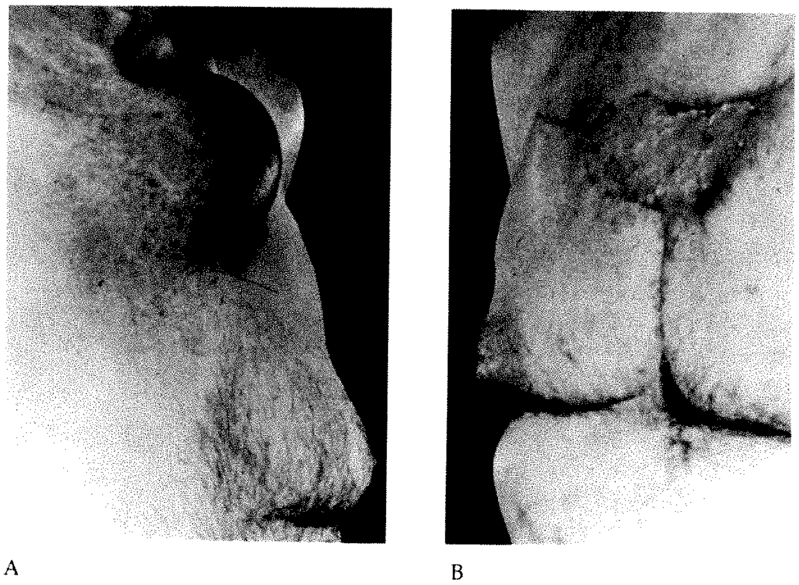

Fig 1. (A) Preoperative view and (B) postoperative view of solitary plasmacytoma involving manubrium.

clonal gammopathy, which can occur as a solitary plasmacytoma of bone or as an extramedullary plasmacytoma [5]. The relationship between multiple myeloma and plasmacytoma has not been fully elucidated [6, 7]. Localized plasmacytomas account for approximately 5% of all plasma cell neoplasms [8]. Multiple myeloma develops in 40% to 60% of patients with solitary plasmacytoma, usually within 10 years of diagnosis [9].

The method of treatment of solitary plasmacytoma depends on the extent of the disease. Minimum evaluation should include bone marrow aspiration, skeletal survey, bone scan, and serum and urine electrophoresis. Twenty-four–hour urine collection for Bence Jones proteins and blood urea nitrogen, creatinine, and serum calcium studies should also be done. For local solitary plasmacytoma, surgery and radiotherapy, either alone or combined, are the principal methods of treatment [4, 10–12]. This includes local surgical removal of the tumor or operation followed by irradiation (usually more than 3,500 rads).

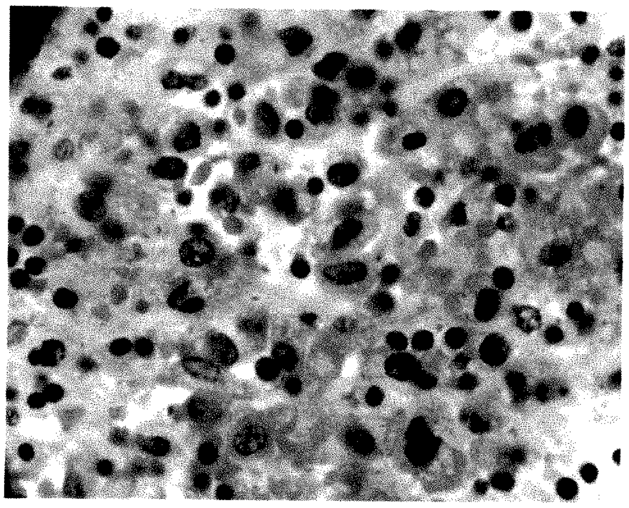

Fig 2. Sheets of immature plasma cells. Nuclear variation is pronounced. Most cells show irregular chromatin pattern. (Hematoxylin and eosin; ×1,024 before 41% reduction.)

There are no large series describing treatment of solitary plasmacytomas involving the ribs or sternum. Most information is gained from reviewing large series of chest wall tumors or representative series of plasmacytomas in general.

Table 1 summarizes several large series of chest wall tumors involving the ribs or sternum. Additional larger series also report involvement of the ribs or sternum. Table 2 summarizes the treatment of myelomas involving the ribs or sternum and the results. In general, the documentation of solitary plasmacytoma versus multiple myeloma is poor. As a result, a true academic statement regarding proper treatment based on a retrospective review cannot be made.

The criteria for diagnosis of solitary plasmacytoma were summarized by Bataille and Sany [9] in a review of 114 cases, and include the following: (1) presence of a solitary bone lesion on the roentgenograms; (2) a biopsy specimen showing plasma cell histology; (3) absence of myeloma cells in the bone marrow aspirate; and (4) absence of anemia, hypercalcemia, and renal involvement. The detection of a monoclonal component in the serum or urine does not exclude the diagnosis of solitary myeloma.

Sixty-nine cases of myeloma involving the chest wall were identified (see Table 2). The sparse details regarding location, method of diagnosis, and exclusion of myeloma are given. Treatment is documented for 6 of the 10 patients in whom the sternum was involved. Five were treated with radiotherapy. Three of them are alive without tumor 28 months to less than 10 years later, and one died with tumor at 4 years. One patient underwent surgical intervention only and is alive at 21 months.

In view of the small number of cases and the absence of exact details regarding size, location, methods of diagnosis, and long-term follow-up, it is suspect to categorically assign patients to radiotherapy alone. Based on our ret-

Table 1. Series of Chest Wall Tumors Involving Ribs or Sternum

Reference	Year	Rib	Chest Wall, Sternum	Myeloma Total	Sternum	Ribs
Hochberg [13]	1953	101	...	3	...	...
Pascuzzi et al [14]	1957	86	18	19	4	15
Watkins and Gerard [15]	1960	37	...	1	...	...
Ochsner et al [16]	1966	36	...	9	...	...
Threlkel and Adkins [17]	1971	23	...	3	...	...
Teitelbaum [18]	1972	340	...	10	...	...
Meyer and Schulz [11]	1974	...	...	1	...	1
Martini et al [3]	1974	...	14	1	1	...
Mill and Griffith [4]	1980	...	...	2	2	...
Paris et al [19]	1980	...	4	1	1	...
Graeber et al [2]	1982	110	2	8	...	...
Sabanathan et al [1]	1985	27	4	8	1	1
Pairolero and Arnold [10]	1985	100	22	2	...	...
Greenberg et al [12]	1987	...	...	1	1	...

rospective review and the success with our 2 patients, serious consideration should be given to en bloc resection of solitary plasmacytoma involving the chest wall. Improved techniques in chest wall reconstruction make this an alternative primary treatment or, at best, an effective debulking procedure to decrease the morbidity of large doses of radiotherapy either preoperatively or postoperatively.

Table 2. Summary of Data on Myelomas Involving the Chest Wall

Reference	Solitary Plasmacytoma	Site	Treatment	Outcome
Hochberg [13]	1	?	Surgery	Died of coronary artery disease at 3 yr
	1	?	Radiotherapy	NED at 10 mo
	1	?	Radiotherapy	Alive and well at 5 mo
Pascuzzi et al [14]	15	Rib	Surgery, 2; local excision, 13; radiotherapy, 6	Died at >5 yr, 5; died at <5 yr, 8; alive and well at >2 yr after operation, 1; alive >8 yr after operation and radiotherapy, 1
	4	Sternum	?	Eventual dissemination, 3
Watkins and Gerard [15]	1	?	Biopsy	Died of general disease at 10 mo
Ochsner et al [16]	9	?	?	?
Threlkel and Adkins [17]	3	?	?	?
Teitelbaum [18]	10	?	?	?
Meyer and Schulz [11]	1	Rib	Surgery	Dissemination at 7 yr; died at 9 yr
Martini et al [3]	1	Sternum	Radiotherapy	Survival >10 yr
Paris et al [19]	1	Sternum	Surgery	Alive and well at 21 mo
Mill and Griffith [4]	1	Sternum	Radiotherapy (3,444 rads)	Died with tumor at >4 yr
	1	Manubrium	Radiotherapy (5,200 rads)	Alive and well at >4 yr; NED
Graeber et al [2]	8	Rib/clavicle	Surgery, chemotherapy, radiotherapy	?
Sabanathan et al [1]	8	Rib, 7 Sternum, 1	Surgery Radiotherapy	Alive and well, 4/8 ?
	1	?	?	?
Pairolero and Arnold [10]	1	?	?	?
Greenberg et al [12]	1	Sternum	Radiotherapy (20 Gy)	NED at 28 mo

NED = no evidence of disease.

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Total Lymphoid Irradiation in Heart Transplantation: Adjunctive Treatment for Recurrent Rejection

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In the face of recurrent heart transplant graft rejection refractory to all conventional immunotherapy, retransplantation is customary treatment. The case of a heart transplant recipient unsuitable for retransplantation whose recurrent rejection was successfully treated with postoperative total lymphoid irradiation is described.

(Ann Thorac Surg 1989;48:863–4)

Although excellent long-term results are achieved with heart transplantation using triple immunosuppression with cyclosporine, prednisone, and azathioprine [1], recurrent rejection refractory to all conventional treatment including pulsed methylprednisolone, antilymphocytic preparations, and OKT3 occasionally occurs. Graft failure or fatal infection usually ensues unless retransplantation is performed. We report the clinical use of postoperative total lymphoid irradiation (TLI) as successful adjunctive treatment for recurrent human heart transplant rejection unresponsive to all conventional immunosuppressive therapy.

A 35-year-old man underwent heart transplantation for the first time at Stanford University in November 1976 for viral cardiomyopathy. He enjoyed excellent functional recovery with only two episodes of rejection, each successfully treated with methylprednisolone and Stanford rabbit antithymocyte globulin.

By March 1987, diffuse graft coronary artery occlusive disease had developed, and he underwent a second orthotopic heart transplantation on June 15, 1987, at Vanderbilt University Medical Center. Retrospective crossmatch was negative; human lymphocyte antigen typing revealed a three-antigen match. Rejection on postoperative day 7 was treated with pulsed methylprednisolone, and two subsequent episodes of rejection were treated with Nashville antithymocyte serum in addition to steroid pulses. Daily maintenance immunotherapy consisted of azathioprine (2 mg/kg), prednisone (0.2 to 0.6 mg/kg), and cyclosporine (5 to 10 mg/kg). The patient continued to demonstrate recurrent episodes of moderate to severe rejection as documented by endomyocardial biopsies.

In response to severe histological rejection and clinical evidence of deteriorating graft function on day 49, a 14-day course of OKT3 (5 mg/day) was begun. Biopsy at the end of treatment documented resolution of rejection. Within four days, rejection recurred. Four additional courses of OKT3 were administered to treat recurrent episodes of rejection. Each episode cleared with treatment but was followed by recurrent rejection within 2 to 3 weeks. Retransplantation was precluded by an unrelenting series of infections.

Total lymphoid irradiation was begun. The patient received 360 rads in four treatments to the inverted Y and mantle using 6-MV photons over 21 days. Total lymphoid irradiation was suspended when meningoencephalitis and leukopenia developed. Episodes of moderate to severe rejection continued. Total lymphoid irradiation was resumed with an additional 1,600 rads given in 20 fractions over 81 days. The last rejection episode occurred in midcourse of the TLI therapy; the patient has been free from rejection for more than a year as documented by bimonthly surveillance biopsies.

Comment

Specific and durable graft tolerance is the goal of transplant immunosuppression. The immunological response to TLI was first documented in patients cured of Hodgkin's disease by radiation therapy [2]. In animal transplant models, TLI has been used to induce suppression of cell-mediated immunity. In 1978, Slavin and colleagues [3] reported the possibility of skin, marrow, and heart tolerance with TLI in a rat model. Myburgh [4], using TLI alone and in combination with other agents in primate models of liver and kidney transplantation, found graft tolerance occurred in a third to a half of the animals when TLI alone was used. Neither bone marrow transplantation nor blood transfusion was essential for tolerance induction.

The Stanford group [5] achieved long-term canine heart allograft survival with preoperative TLI and postoperative antithymocyte globulin and azathioprine. In rat heart allotransplantation, TLI in combination with low-dose cyclosporine was reported to be synergistic [6], and in a

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hamster to rat xenograft model, TLI with cyclosporine permitted successful long-term survival [7]. The combination of TLI and antithymocyte globulin without bone marrow transplantation in a canine heart allograft transplant model was found to be more effective in preventing rejection than either TLI alone or TLI in combination with other agents [8].

In human studies of cadaveric kidney transplants, Saper and associates [9] demonstrated that TLI effectively reduced the requirements for immunosuppressive drugs. They showed that TLI caused T-cell lymphocytopenia. The relative proportions of T-lymphocyte subsets were altered, with an increase in suppressor-cytotoxic T-cells (Leu 2+, T8) and a decrease in helper-inducer T-cells (Leu 3+, T4). Similarly in our patient, T4 fell to 18% (normal, 36% to 60%) and T8 increased to 57% (normal, 17% to 43%) with suppression of the T4/T8 ratio during the course of TLI.

Reports of TLI in human heart transplantation are limited. Preoperative TLI in combination with donor bone marrow transplantation was reported in 6 heart transplant patients [10]. Two were alive at 4 years, 1 died of heart failure at 10 days, 2 died of infection within 7 weeks, and 1 died of rejection at 7 months. There have been no previous reports of postoperative TLI used to treat recurrent heart transplant rejection.

In summary, this case report demonstrates that TLI can be used successfully as adjunctive treatment of refractory cardiac rejection unresponsive to conventional therapy. Further studies in controlled prospective trials will be necessary to determine the overall efficacy of this mode of immunosuppressive therapy.

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Carotid and Aortic Arch Endarterectomy Using Hypothermic Arrest With Coronary Bypass

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A 67-year-old man with symptomatic bilateral carotid artery obstructions and a large, friable atheromatous plaque of the transverse aortic arch required coronary artery bypass grafting for severe triple-vessel disease. An endarterectomy of the transverse arch and a left carotid endarterectomy were performed using deep hypothermic circulatory arrest concomitant with quadruple coronary artery bypass grafting. Recovery was uneventful. Hypothermic circulatory arrest provides adequate protection for this combined procedure and may eliminate cerebral embolization.

(Ann Thorac Surg 1989;48:865–6)

Stroke caused by atherosclerosis is a devastating complication of open heart operations. Atherosclerosis of the carotid artery, the ascending aorta, or the transverse arch has been defined as a major risk factor for perioperative stroke, and these can commonly coexist. In 1986, Culliford and associates [1] proposed the technique of visual inspection of the aorta with removal of hazardous debris under deep hypothermic circulatory arrest using central aortic cannulation. We successfully applied their technique with a concurrent carotid endarterectomy, but used femoral artery cannulation.

A 67-year-old man with a history of right eye amaurosis fugax and global cerebral ischemic symptoms with a 2-month history of angina pectoris underwent cardiac and carotid artery evaluation. On physical examination, a grade 2/6 left carotid bruit was noted. A treadmill test was positive with 2-mm ST segment depression at two minutes.

Coronary angiography demonstrated severe triple-vessel disease with 100% obstruction of the left anterior descending coronary artery, 90% obstruction of the obtuse marginal branch and the circumflex coronary artery, and 100% obstruction of the distal right coronary artery with an ejection fraction of 40%. Digital subtraction angiography confirmed total occlusion of the right internal carotid artery and 85% stenosis of the left internal carotid artery with crossfilling to the right cerebral hemisphere. In addition, the patient had a large, elevated-appearing plaque within the aortic arch (Fig 1). This appeared to narrow the arch about 40%.

Because of multivessel coronary artery disease coexisting with symptomatic bilateral carotid artery disease and severe atherosclerosis of the transverse arch, simultaneous surgical intervention for the three lesions was planned. At operation, the left carotid artery was exposed first, and then a median sternotomy incision was performed. After arterial cannulation of the left common femoral artery and venous cannulation of the right atrium, cardiopulmonary bypass was initiated. The nasopharyngeal temperature was lowered to 16°C during 40 minutes of perfusion. During this cooling period, no manipulation of the aorta was allowed.

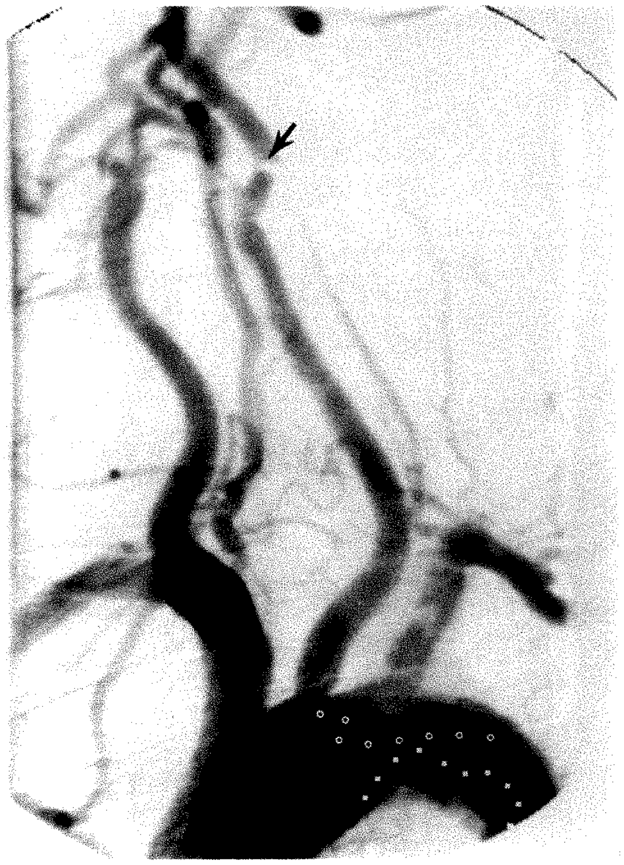

*Fig 1. Digital subtraction angiograph showing a large, elevated-appearing plaque in the aortic arch (*white dots and squares*) and 85% stenosis of the left internal carotid artery (*black arrow*).*

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When cooling was complete, total circulatory arrest was introduced, and the left carotid endarterectomy was performed. After a clamp had been placed on the innominate artery, a long incision was made along the inner curvature of the transverse arch. Exploration of the arch revealed a thick, soft plaque measuring 4 × 8 cm. This friable debris was scraped and wiped out, and then the arch was irrigated clean. Before closure of the aorta, cardiopulmonary bypass was reinstituted, and the aorta was flushed retrograde before completion of the aortic closure. The clamp on the innominate artery was then released, and the left common carotid artery was backbled before its closure was completed. The body temperature was rewarmed to 30°C and maintained.

During the slow rewarming period, the aorta was cross-clamped, and cold cardioplegic solution was given. Quadruple saphenous vein bypass grafting to the diagonal, left anterior descending, obtuse marginal, and posterior descending arteries was performed. Circulatory arrest time was 31 minutes and aortic cross-clamp time, 40 minutes; thus, total time of ischemia for the heart was 71 minutes. The total pump time was 142 minutes. Throughout the perfusion period, a flow rate of 2.4 L/min/m^2 and a mean perfusion pressure ranging between 60 and 80 mm Hg were maintained.

The patient's postoperative course was uneventful; in particular, there were no neurological complications. He was discharged on the tenth postoperative day.

Comment

Hertzer and associates [2] observed a 7.35% incidence of stroke in patients with severe bilateral carotid artery stenosis who underwent coronary artery bypass grafting compared with a 1.8% incidence for the total population of patients having bypass grafting. Isolated carotid endarterectomy performed in the presence of severe coronary artery disease has been associated with a high mortality of 20% from myocardial infarction, and the mortality for staged carotid and coronary artery operation was high compared with that for the simultaneous procedure [3].

The simultaneous carotid and coronary artery operation has been indicated when bilateral or symptomatic carotid artery disease or a combination of these is associated with unstable angina, left main obstruction, or multivessel disease [4]. Gardner and colleagues [5] identified severe atherosclerosis of the ascending aorta as one of the major risk factors for stroke after coronary artery bypass grafting. The incidence of stroke was significantly higher in that group compared with the incidence in the control group (14% versus 3%; $p < 0.005$).

Several techniques have been reported to minimize this risk. These include the use of an intraaortic Foley balloon catheter [6] and "no-touch" methods such as innominate–coronary artery bypass grafting [7] or coronary–coronary artery bypass grafting [8]. Even if these indirect methods are adopted, the risk of embolization caused by manipulation of the aorta or the "sandblast" effect of the arterial cannula on friable plaques is still present.

Culliford and associates [1] introduced a visual, aggressive technique, which consists of direct inspection of the diseased aorta with removal of hazardous debris under deep hypothermic circulatory arrest. They used selected cannulation of the ascending aorta or the transverse arch with a long cannula. We adopted femoral artery cannulation instead because cannulation of the ascending aorta through the transverse arch might have caused dislodgment of the plaque. Combining both the carotid endarterectomy and the transverse arch endarterectomy using deep hypothermic arrest at the time of coronary artery bypass grafting in this patient was safe and successful, and prevented cerebral embolization.

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The Serratus Sling: A Simplified Serratus-Sparing Technique

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A partial muscle-sparing technique is presented that combines the advantage of muscle sparing with the wide exposure offered by a posterolateral incision. The technique is simple and quick, does not require elaborate equipment, and facilitates thoracotomy closure.

(Ann Thorac Surg 1989;48:867–8)

The serratus sling technique is a modification of the standard posterolateral thoracotomy. It was developed to combine the wide exposure offered by a posterolateral incision with the advantages of muscle sparing.

Technique

The patient is positioned on the operating table as for a standard posterolateral thoracotomy. An incision is made that extends from the anterior border of the latissimus dorsi, continues horizontally across the lateral chest passing 2 to 3 cm below the scapular tip, and turns cephalad posteriorly midway between the spine and posterior scapular edge. Posteriorly, the incision is stopped when the trapezius muscle edge is exposed. The latissimus dorsi muscle is divided using electrocautery and the posterior edge of the divided muscle is mobilized superiorly and inferiorly to allow for a two-layer closure (Fig 1).

Continuous with the serratus, the fatty tissue of the ausculatory triangle is divided posteriorly to the rhomboid and anteriorly to free the lateral edge of the serratus. The bony chest wall is thus exposed laterally; however, the unretracted serratus covers the medial third to half of the incision. The long thoracic neurovascular bundle is identified lying superficially on the serratus. A Kelly clamp is placed through the serratus muscle medial to the long thoracic nerve and a large Penrose drain is grasped (Fig 2). The Penrose drain, which encompasses the lateral border of the muscle and its neurovascular bundle, is retracted medially as a serratus sling (Fig 3). The ribs are handled as per the surgeon's routine. If further exposure is necessary, it is obtained posteriorly, dividing portions of the trapezius and rhomboid muscles. We have found this to be necessary infrequently. The serratus is never divided.

Closure involves rib reapproximation as per routine. The serratus sling is released and the divided fatty layer is closed with a running 1-0 Vicryl suture. The latissimus dorsi muscle is closed in 2 layers with 1-0 Vicryl sutures; a subcutaneous and subcuticular skin stitch completes the closure.

Comment

The importance of preserving muscle function in performing thoracotomy incisions is generally accepted and was stressed by Alley [1]. Recently, additional reports have described muscle-sparing modifications of the "standard" axillary [2], anterolateral [3], lateral [4], and posterolateral [5, 6] thoracotomies. The theoretical advantages of a muscle-sparing technique include more rapid recovery of respiratory function and arm and shoulder mobility, less severe postoperative pain, a better seal because multiple incisions do not overlie one another, and quicker closure. Additionally, an undivided muscle may be used subsequently for a flap. Most muscle-sparing techniques, however, trade muscle preservation for exposure. Our modification, we believe, does not.

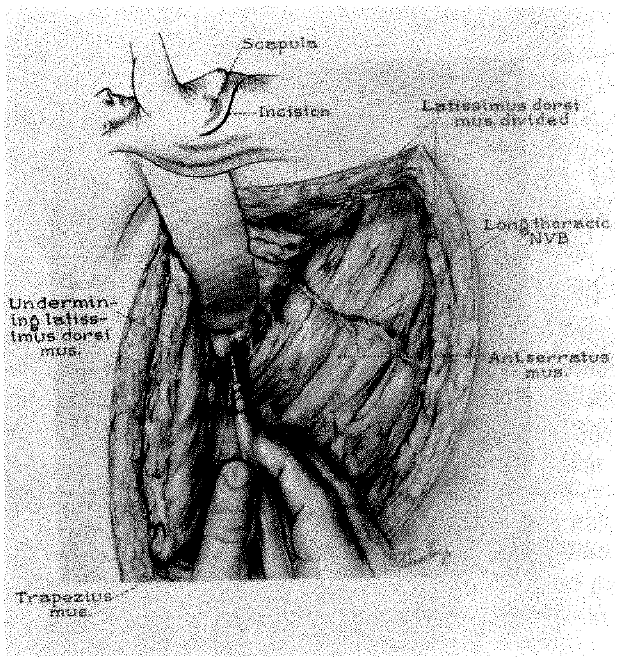

Fig 1. The latissimus dorsi muscle has been divided and its posterior edge is mobilized. (Ant. = *anterior;* mus. = *muscle;* NVB = *neurovascular bundle.)*

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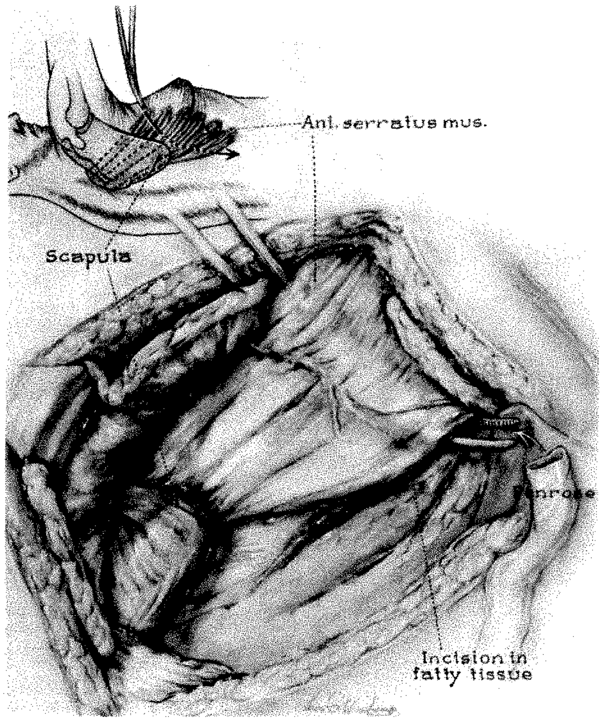

Fig 2. A Penrose drain is passed around the lateral edge of the serratus; the neurovascular bundle is encompassed and preserved. (Ant. = *anterior;* mus. = *muscle.)*

The standard posterolateral thoracotomy involves dividing all or substantial portions of four muscle groups—the latissimus dorsi, trapezius, serratus anterior, and rhomboid major [7]. By the present modification of the posterolateral incision, the latissimus dorsi is the only muscle routinely divided. The serratus is always preserved using a Penrose sling technique, and the trapezius and rhomboid are infrequently divided, and then only partially as needed. Despite sparing the above muscles, the advantages of the standard incision, namely, wide exposure and easy posterior extension, are preserved. This is, in fact, a modified posterolateral incision and not a lateral thoracotomy. A lateral thoracotomy may begin anteriorly in the inframammary fold but does not extend posterior to the lateral border of the latissimus dorsi, regardless of whether the muscle is preserved or divided [8]. A posterolateral incision continues cephalad posteriorly, exposing a part of the trapezius and rhomboid muscles, as with this incision.

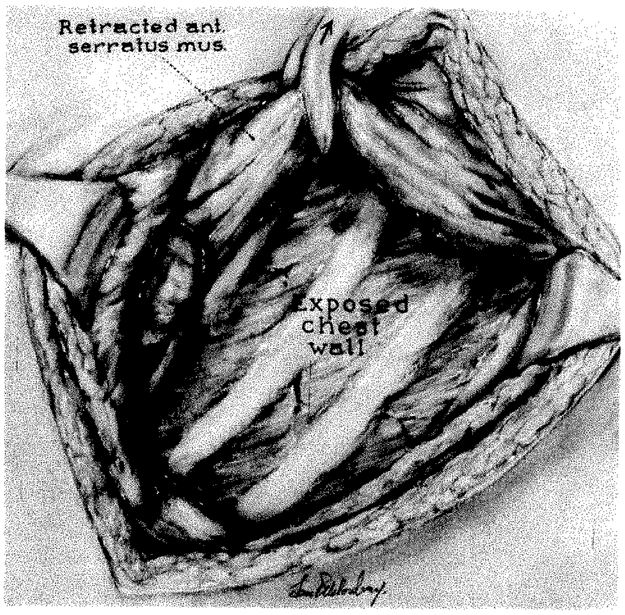

Fig 3. Chest wall exposure with the serratus sling. (ant. = *anterior;* mus. = *muscle.)*

Partial muscle-sparing modifications of the standard posterolateral incision are not new. Many techniques spare the serratus by leaving it in situ (this is particularly cumbersome for the first assistant), retracting it by hand medially, or retracting it by modified chest spreaders. The technique described in this report does not require help with retraction, elaborate equipment, or additional time for its use beyond that required by the standard posterolateral thoracotomy.

The serratus anterior reportedly does not contribute to respiratory function but stabilizes and rotates the scapula for full arm and shoulder range of motion. Nonetheless, the long thoracic nerve that enervates it was originally called the external respiratory nerve of Bell [9]. Whether preserving the serratus enhances return of postoperative respiratory function is conjectural, but it does appear to facilitate earlier full arm and shoulder mobility, in addition to the other previously stated advantages of a muscle-sparing approach. We now use this technique as our standard posterolateral thoracotomy.

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Improved Visualization of the Internal Mammary Artery With a New Retractor System

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A new sternal retractor system was developed to improve exposure of the internal mammary artery and protect the lung. This retractor can be used for either single or bilateral dissection of the internal mammary artery. It has been used in more than 2,000 cases with excellent results.

(Ann Thorac Surg 1989;48:869–70)

Dissection of internal mammary artery (IMA) pedicles can be tedious and awkward. Internal mammary artery grafts are being used routinely, and good technique is important to achieve consistently good results. A new retractor system was developed (Omni-Tract Surgical, St. Paul, MN) that greatly improves the technique of developing an IMA pedicle.

Description

Several features of this sternal retractor make it superior to other systems. Visualization of the IMA pedicle is greatly improved with the fiberoptic light source, lung retractor, and swivel rakes (Fig 1). The self-retaining cool fiberoptic light source greatly increases the intensity of the light in the area of the surgeon's choice without the need to move overhead lights or wear headlights. The self-retaining, malleable lung retractor allows depression of the lung to prevent thermal injury to it and keeps the surgeon's line of sight clear, which is especially helpful with emphysematous lung. The swivel rakes allow for a greater purchase area on the sternum and thus minimize damage to the sternum.

For ease in set up and decreased risk of contamination of the field a clamp attachment and split post assembly are used (Fig 2). The clamp allows fixation of this unit in the sterile field without requiring assistance from a circulating nurse. The split post assembly allows for transfer of the

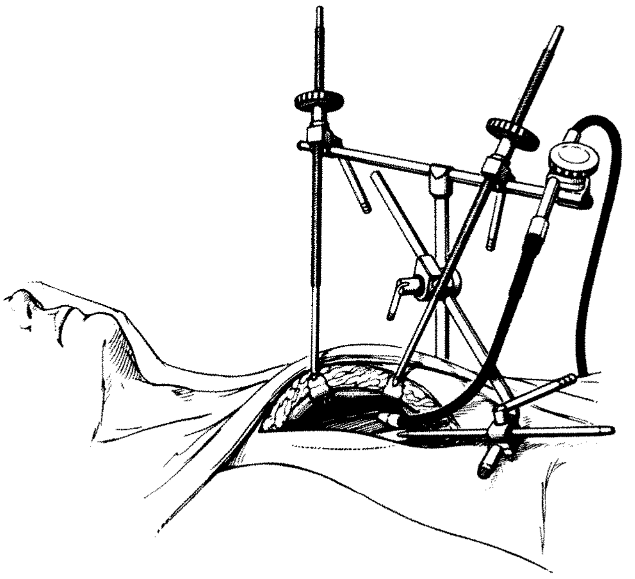

Fig 1. Complete assembly in position.

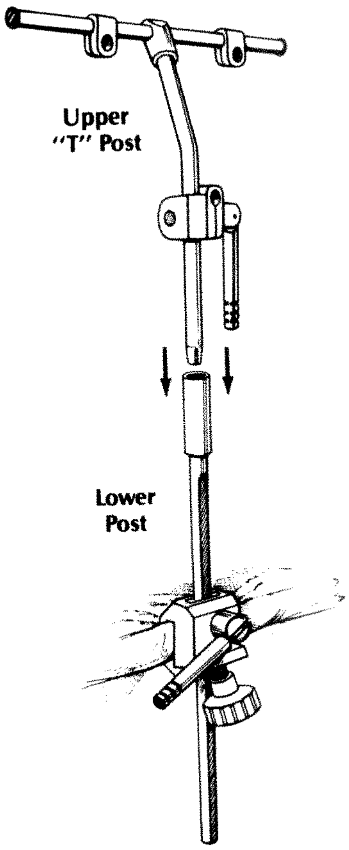

Fig 2. Detail of clamp mechanism.

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upper post across the table in the case of a bilateral IMA dissection to a second, lower post on the opposite side.

Results

This retractor system was adopted in 1986 as the primary retractor system for IMA operations and has been used in more than 2,000 cases. It saves considerable time and provides a better-quality IMA with less strain on the operating surgeon.

Conclusion

Reproducible exposure with good lighting and without damage to the retracted tissues is essential to good operative technique. This retractor system has been valuable in providing these conditions, which are especially important now that almost every bypass patient receives an IMA graft because of its long-term patency.

Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. They should also keep a record of their attendance at thoracic surgical meetings, and other continuing medical education activities, for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS III syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS III booklets prior to applying for recertification. SESATS III booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1992 may begin the recertification process in 1990. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1990. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Left Thoracotomy Approach for Coronary Artery Bypass Grafting in Patients With Pericardial Adhesions

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The left thoracotomy approach represents a safe alternative to median sternotomy for coronary artery bypass grafting in patients with pericardial adhesions. It provides an excellent exposure to the lateral aspect of the heart, and isolated circumflex coronary revascularization can advantageously be performed via this approach in select cases.

(Ann Thorac Surg 1989;48:871–3)

Coronary bypass reoperations are routinely performed through a standard median sternotomy. Adhesions impose an important technical difficulty at resternotomy, however; damage to the heart or coronary grafts during sternal reentry may lead to perioperative myocardial infarction and death [1], and mobilization of the heart from adhesions increases bleeding complications and may be a source of embolization of debris from atherosclerotic vein grafts.

The left thoracotomy approach represents a safe alternative to repeat sternotomy for coronary reoperations [2, 3]. Anterolateral left thoracotomy provides a good exposure to the lateral aspect of the left ventricle and gives direct access to the circumflex coronary arterial system. Minimal intrapericardial dissection is required, and the risk of injury to coronary grafts (patent internal mammary artery implants) is reduced.

Technique

The patient is placed in the right lateral decubitus position. The vein is harvested from either the right or the left leg, and the left femoral vessels are exposed for cannulation (Fig 1).

Simultaneously, the chest is entered through a large anterolateral thoracotomy through the fourth intercostal space. The pericardium is opened parallel and anterior to the phrenic nerve (Fig 2). After minimal dissection of pericardial adhesions, an excellent exposure to the lateral aspect of the heart can be obtained.

Femorofemoral bypass is then instituted. The main pulmonary artery can be cannulated to increase venous drainage. The patient is progressively cooled to 24°C. At the onset of ventricular fibrillation, the heart is unloaded through the left atrial appendage (Fig 3).

The circumflex coronary vessels are easily exposed: the stem of the circumflex coronary artery and its obtuse marginal and posterolateral branches can be grafted via this approach. However, access to the left anterior descending coronary artery and the right coronary arterial system is poor.

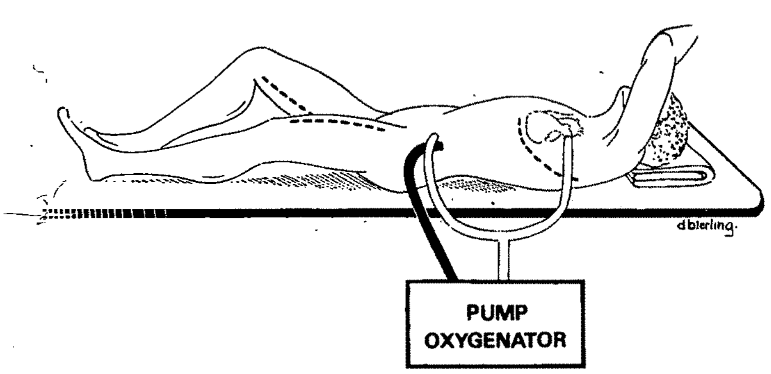

Fig 1. Positioning of the patient. Femorofemoral bypass is instituted; additional venous drainage can be obtained by cannulation of the main pulmonary artery.

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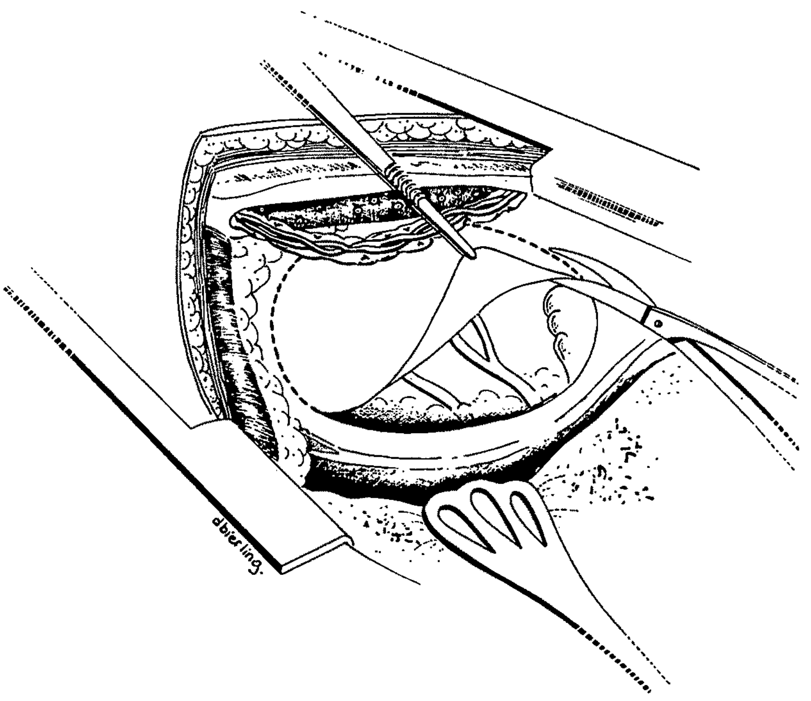
Fig 2. The pericardium is incised anterior to the phrenic nerve, and circumflex coronary vessels are exposed.

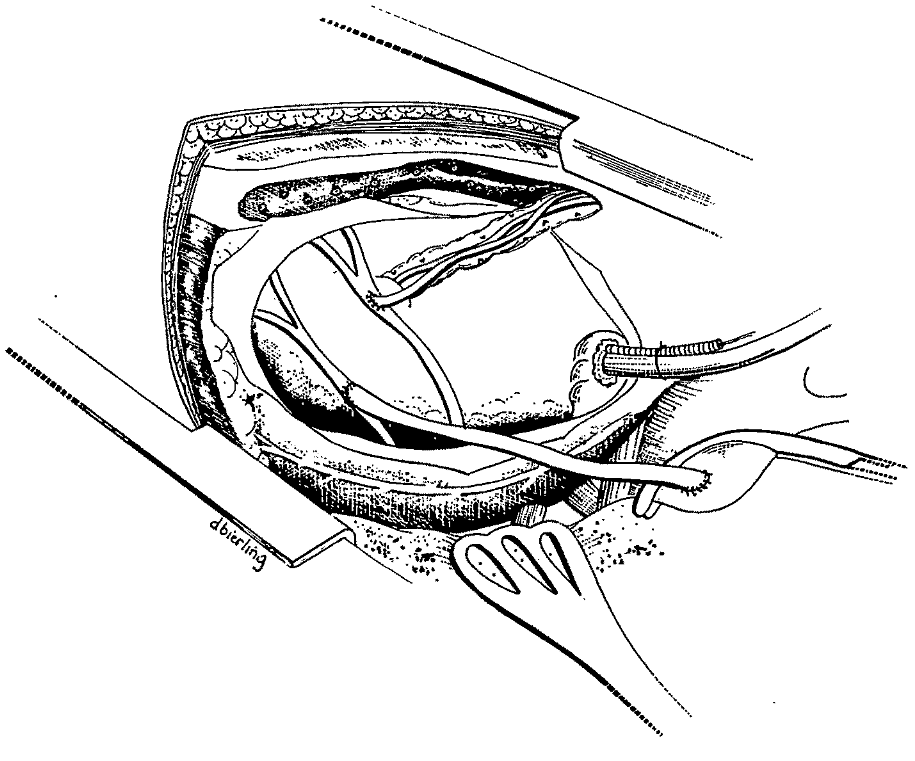


After arteriotomy, stay sutures are placed to obtain a dry surgical field. Distal anastomoses are then completed; the marginal and posterolateral branches are advantageously grafted in a sequential fashion with the same venous conduit.

Internal mammary artery grafting is possible through anterolateral thoracotomy. The left internal mammary artery can easily be harvested all the way to its origin on the subclavian artery by this approach (Fig 2); however, one should be careful not to injure the phrenic nerve in dissecting the proximal implantation of this vessel.

Proximal anastomoses of the venous conduits are made to the aortic isthmus with side clamping; this provides the grafts a direct and harmonious course to the aorta. Proximal anastomosis may also be performed to the descending aorta [4, 5] or to the left subclavian artery [2, 3]. At the

Fig 3. Distal anastomoses are completed after the heart is unloaded through the left atrial appendage. Proximal anastomosis is performed to the aortic isthmus using side-clamping.



end of the operation, the pericardium is not closed, and the left pleural cavity is drained with one large chest tube.

Comment

Since 1982, coronary artery bypass grafting was performed via an anterolateral left thoracotomy in 5 patients with pericardial adhesions in our institution. Three of these patients had undergone previous coronary operations 5, 7, and 8 years earlier, and in 1 patient coronary revascularization had already been achieved on two occasions (6 years and 1 year earlier, respectively). In the fifth patient, pericardial adhesions had resulted from mediastinal irradiation for lymphoma. All 4 patients undergoing coronary reoperation had a patent graft to the left anterior descending coronary artery (two internal mammary arteries and two venous grafts).

Circumflex coronary revascularization was realized on the following sites: obtuse marginal branch (n = 5 including one internal mammary artery graft), posterolateral branch (n = 2), and stem of the circumflex (n = 1). Additional saphenous vein grafts to other vessels were performed in 2 patients (left anterior descending artery [n = 1] and left ventricular branch of the right coronary artery [n = 1]). All patients survived and had an uncomplicated postoperative recovery, although 1 patient required temporary assistance by intraaortic balloon pumping.

We believe that anterolateral left thoracotomy is a valuable approach for coronary revascularization in select patients. It represents an advantageous alternative to sternotomy in the following situations: (1) need for isolated circumflex coronary bypass grafting in patients with pericardial adhesions due to previous coronary operation or mediastinal irradiation and (2) patent internal mammary artery or vein graft constructed on the left anterior descending coronary artery that might be injured during sternal reentry.

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Axillary Artery Insertion of an Intraaortic Balloon Pump

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We describe a technique for insertion of an intraaortic balloon pump through the axillary artery. This approach is useful in patients requiring intraaortic balloon pump support before cardiac transplantation in that it dramatically increases patient mobility while awaiting a donor heart.

(Ann Thorac Surg 1989;48:874–5)

The use of intraaortic balloons (IABs) to bridge patients to cardiac transplantation is well established [1, 2]. Unfortunately, a shortage of donor hearts frequently results in a prolonged period of mechanical support before transplantation. The standard insertion technique through the femoral artery severely limits the patient's mobility and results in confinement to bed. As a result, these already compromised patients are at a high risk for the development of pulmonary complications including pneumonia and embolism, which may preclude transplantation. Insertion of an IAB through the iliac artery improves patient mobility and may allow ambulation; however, it requires a retroperitoneal approach for access to the vessels [3]. Two case reports [4, 5] of IAB insertion through the subclavian artery have demonstrated the feasibility of using an upper extremity artery for access to the aorta. The subclavian artery was exposed by resecting the right clavicle in one report and through a left supraclavicular incision in the other. Intraaortic balloons were positioned in the descending aorta through grafts anastomosed to the subclavian artery. Both patients were successfully augmented; however, patient mobility was not described. In an attempt to improve the mobility of our patients who were being bridged to cardiac transplantation, we developed a technique of IAB insertion through the axillary artery. The procedure can be performed under local or general anesthesia without the use of a graft and allows the patient full mobility.

Technique

The left or right axillary artery is exposed through a transverse infraclavicular incision extending from the deltopectoral groove to the midclavicle. The pectoralis major is split in the direction of its fibers. The clavipectoral fascia is divided, exposing the axillary artery and vein as well as the brachial plexus. The vein is gently retracted and circumferential control of the artery is obtained by sharp dissection. Care is taken not to disrupt any of the arterial branches that may provide collateral circulation. A 4-0 polypropylene horizontal mattress stitch is placed in the vessel wall. With use of the Seldinger technique, a 0.03-in (0.76 mm) guidewire is positioned under fluoroscopic guidance into the descending aorta. If the guidewire cannot be manipulated into the descending aorta, an 8F multipurpose coronary angiography catheter is used to direct the guidewire distally from the origin of the subclavian artery. A 9.5F double-lumen IAB (Datascope, Paramus, NJ) is inserted through a second small skin incision at the lateral pectoral margin and tunneled subcutaneously into the infraclavicular incision. The balloon catheter is then passed over the guidewire and positioned in the descending aorta so that the most proximal portion of the balloon is located just beyond the origin of the left subclavian artery. In general this results in the distal end of the IAB being located at the second or third lumbar vertebral body (Fig 1). Fluoroscopy and arterial pressure are used to check IAB position and augmentation.

The management of an axillary IAB is identical to that of transfemoral IABs. Anticoagulation therapy consists of intravenous dextran sulfate or heparin. With the axillary insertion, the waveform is monitored using the distal port on the balloon, and counterpulsation is timed in a fashion identical to that employed with a transfemoral placement. Although arterial lines placed in the contralateral arm or either femoral artery accurately display the identical waveform, we do not use such lines after IAB placement because they may impair ambulation and patient mobility. Because of the partial obstruction of the axillary artery proximal to the site of insertion, radial artery monitoring on the side of IAB placement will likely be inaccurate and should not be employed.

Balloon removal is usually accomplished in the operating room. The incision is reopened and proximal and distal control are again obtained. After complete balloon deflation, the IAB is withdrawn through the arteriotomy and vascular clamps are applied proximal and distal to the site. Each clamp is briefly removed in turn to flush any intraarterial thrombus out through the arteriotomy. The artery is then closed with a 5-0 polypropylene suture. No complications have been encountered when using this approach.

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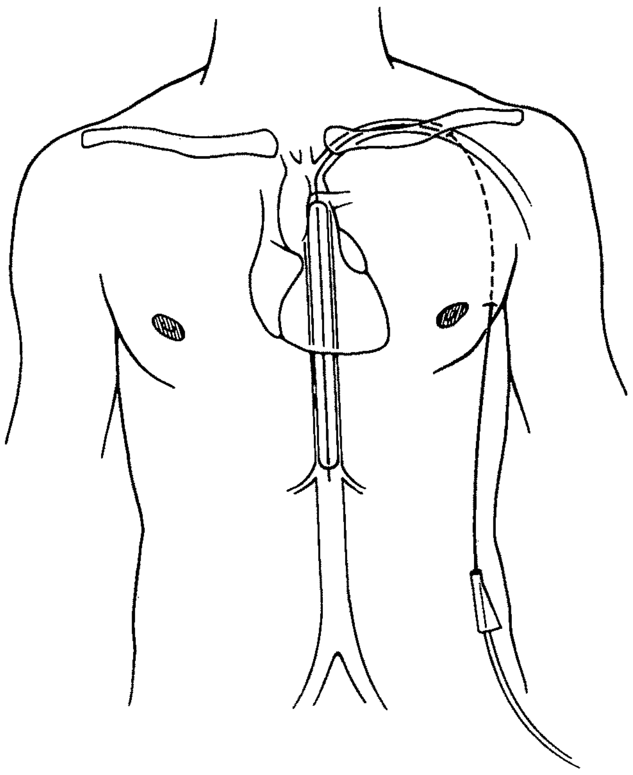

Fig 1. Intraaortic balloon pump positioned in the descending aorta.

Comment

A shortage of donor hearts has resulted in a longer waiting period for all potential heart recipients. Patients requiring IAB counterpulsion before transplantation are severely limited in mobility if the device is inserted through the femoral artery. This predisposes them to the development of complications such as pneumonia and pulmonary embolism, which may preclude transplantation. Transaxillary IAB insertion is easily performed and allows increased patient mobility and ambulation.

This approach might also prove useful in some patients with severe peripheral vascular disease who require IAB support after a cardiac operation. In such patients it may be impossible to pass an IAB or even a guidewire retrograde from a femoral artery. When faced with this situation, an alternative approach is to place an IAB directly through the ascending aorta. This insertion method may be difficult or even dangerous in patients with an ascending aorta that is crowded with multiple proximal anastomoses or with an aorta that is extensively calcified. In such patients an axillary approach may be a safer alternative. In this operative setting digital manipulation of the subclavian artery and aorta can be used instead of fluoroscopy to position the IAB in the descending aorta. The transaxillary approach has the additional advantage of not requiring reopening the sternotomy to effect balloon removal.

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Heller's Contribution to the Surgical Treatment of Achalasia of the Esophagus

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Ernst Heller's original 1914 report of extramucosal esophagomyotomy for the treatment of "cardiospasm" (achalasia) of the esophagus is reviewed in historical perspective, defining those factors that may have contributed to delay in recognition of its true value as the major contribution to the modern management not only of achalasia but also of many other esophageal motor disorders. Special attention is also given to what Heller actually described, observed, and did in the light of current pathophysiological understanding. Finally, emphasis is placed on current management as it has evolved to the present state of technical performance.

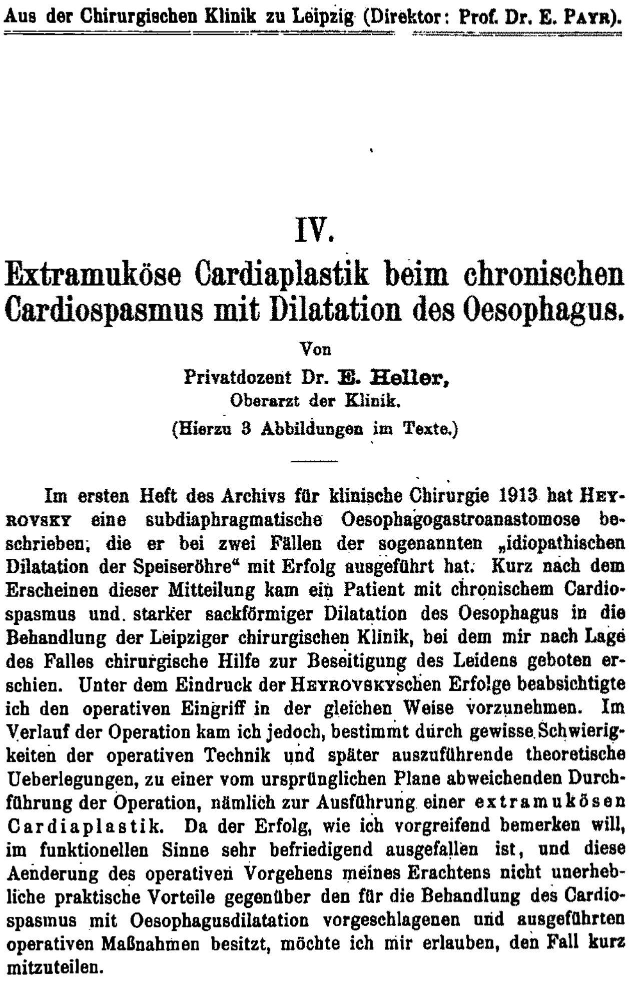
(Ann Thorac Surg 1989;48:876–81)

When he made his famous contribution to the surgical treatment of achalasia (Fig 1), Ernst Heller (Fig 2) was a 36-year-old senior surgical assistant (Oberarzt) to Professor E. Payr* at the University Surgical Clinic in Leipzig, Germany [1, 2].

Although Heller's extramucosal esophagomyotomy (with modification) has become the preferred surgical procedure for achalasia in most parts of the world, it was not initially widely accepted or employed even in his own country. Throughout his career, Heller continued to use the double (anteroposterior) esophagomyotomy of his original description [3]. This was modified in 1918 by De Bruïne Groeneveldt [4] of Holland, who limited the procedure to a single anterior myotomy. Zaaijer [5] is generally credited with having popularized this single-myotomy modification, which, under his influence, was widely adopted throughout most of continental Europe, but not Britain [6] or North America [7].

Although reports of Heller's procedure were never to completely disappear from the German-language literature, the anastomotic cardioplastic procedures that totally destroyed or bypassed the lower esophageal sphincter (Fig 3) continued to be championed in Germany after World War I by the influential Sauerbruch [11] and his protégé Frey [12]. The fact that these cardioplastic procedures negated the sphincteral barrier to gastroesophageal reflux was not immediately apparent, for the cardioplasties were highly effective in relieving the most compelling symptoms of achalasia, namely, esophageal obstruction and retention.

Acceptance of myotomy was more delayed in Britain [6], where the late complications of the anastomotic cardioplasties were not widely appreciated until the classic report of Barrett and Franklin [13]. In North America, supportive clinical and laboratory studies such as those by Ripley and associates [14, 15] finally put the cardioplasty controversies to rest. Thus, it was nearly 40 years after


Aus der Chirurgischen Klinik zu Leipzig (Direktor: Prof. Dr. E. PAYR).

IV.

Extramuköse Cardiaplastik beim chronischen Cardiospasmus mit Dilatation des Oesophagus.

Von

Privatdozent Dr. **E. Heller,**
Oberarzt der Klinik.
(Hierzu 3 Abbildungen im Texte.)

Im ersten Heft des Archivs für klinische Chirurgie 1913 hat HEYROVSKY eine subdiaphragmatische Oesophagogastroanastomose beschrieben, die er bei zwei Fällen der sogenannten „idiopathischen Dilatation der Speiseröhre“ mit Erfolg ausgeführt hat. Kurz nach dem Erscheinen dieser Mitteilung kam ein Patient mit chronischem Cardiospasmus und starker sackförmiger Dilatation des Oesophagus in die Behandlung der Leipziger chirurgischen Klinik, bei dem mir nach Lage des Falles chirurgische Hilfe zur Beseitigung des Leidens geboten erschien. Unter dem Eindruck der HEYROVSKYschen Erfolge beabsichtigte ich den operativen Eingriff in der gleichen Weise vorzunehmen. Im Verlauf der Operation kam ich jedoch, bestimmt durch gewisse Schwierigkeiten der operativen Technik und später auszuführende theoretische Ueberlegungen, zu einer vom ursprünglichen Plane abweichenden Durchführung der Operation, nämlich zur Ausführung einer extramukösen Cardiaplastik. Da der Erfolg, wie ich vorgreifend bemerken will, im funktionellen Sinne sehr befriedigend ausgefallen ist, und diese Aenderung des operativen Vorgehens meines Erachtens nicht unerhebliche praktische Vorteile gegenüber den für die Behandlung des Cardiospasmus mit Oesophagusdilatation vorgeschlagenen und ausgeführten operativen Maßnahmen besitzt, möchte ich mir erlauben, den Fall kurz mitzuteilen.

Fig 1. Title page of Heller's original article as it appeared in Mitteiluungen aus den Grenzgebieten der Medizin und Chirurgie *in 1914 [2].*

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Quotations from Dr Heller are in translation by Maureen J. S. Payne, MA.

* Professor Erwin Payr (1871–1946), Director, University Surgical Clinic, Leipzig; best remembered for the Payr clamp, a crushing clamp used in resection of stomach and intestine.

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Fig 2. Professor Ernst Heller (1877–1964). (From Steichen and Ravitch [1]. Reprinted by permission from the New York State Journal of Medicine, *copyright by the Medical Society of the State of New York.)*

Heller's initial publication that esophagomyotomy was introduced to the surgical practice of North America [16]. At that time, the Heller procedure held the promise of weakening the lower esophageal sphincter sufficiently to prevent obstruction to swallowing but not so completely weakening the sphincter as to allow damaging gastroesophageal reflux.

Undoubtedly, the enormous authoritarian influence exerted by the German Geheimrat professorial system of the early 1900s contributed initially to the fact that the contribution of one so young and academically junior as Heller was ignored in favor of the more expedient techniques espoused by the great German professors of the time. The system was so influential that it virtually dominated surgical opinion throughout Germany and most of the world [17].

Although Heller was named an associate professor of surgery in 1914, his academic career was interrupted from 1914 to 1918 by World War I, in which he served as a battlefield physician. After the war, he returned to Leipzig as surgeon-in-chief at the St. Georg County Hospital. It was not until after World War II that the importance of his contributions was acknowledged, and in 1949, he was named a full professor of surgery at the University of Leipzig [1].

Although Heller was denied immediate recognition for his important contribution, recognition eventually came during his lifetime, which was long and successful. He was to write approximately 80 scientific articles, including ongoing commentary on the technical [18–21] and historical [3, 22–24] aspects of achalasia of the esophagus. Professor Ernst Heller died in Leipzig on November 2, 1964, at the age of 87 years [1].

Heller's 1914 Report

The Patient

Heller's 1914 report [2] centered entirely on the case history of a single patient: a 49-year-old man with a 30-year history of swallowing difficulties. The patient was seen by Heller for an intolerance to solids and a diet largely restricted to liquid or pureed foods. Eating had become a slow and laborious process because copious liquids were required to wash food through the esophagus. Regurgitation of undigested food occurred from one hour to ten hours after meals. Hyperextension of the torso seemed to facilitate the passage of food into the stomach. In these ways, the patient had been able to maintain a marginal nutritional state through the years. Apparently, consultation with Heller was occasioned by an acute near-asphyxiation precipitated by impaction of food in the pharynx when the patient attempted to swallow a large piece of meat. Successful extraction with forceps had saved the patient's life and convinced him of the necessity of definitive resolution of his condition.

On examination, the patient appeared healthy, though thin, and had a notably foul breath. An attempt was made to pass a bougie down the esophagus, but this induced gagging and emesis of what was identified as fresh, undigested food from the previous day. With reintroduction of the flexible rod, resistance to its advancement was encountered at the anticipated level of the lower esophageal sphincter, 42 to 44 cm from the incisor teeth. Resis-

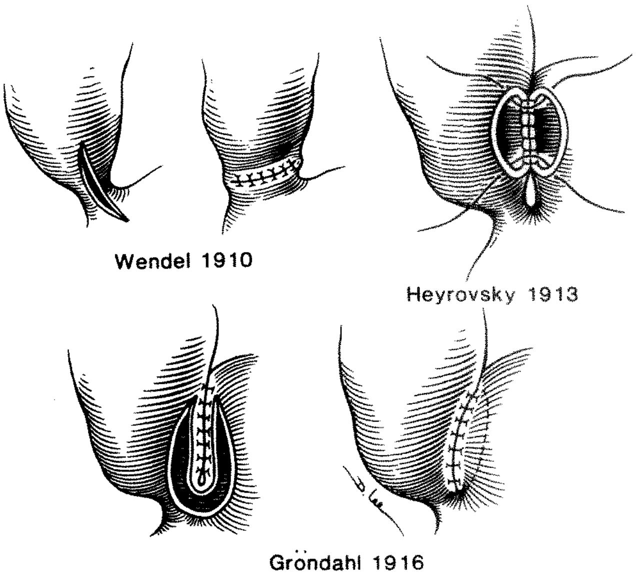

Fig 3. Anastomotic cardioplasties for achalasia [8–10], which were championed by the great German surgeons of the first half of the 20th century. So influential were they that the importance of Heller's contribution was lost for nearly 40 years. Note that these cardioplasties resemble the earlier described pyloroplasties, especially those described by Heineke-Mikulicz (1886–1988), Jaboulay (1892), and Finney (1902). The late devastating reflux consequences of these cardioplastic operations were not widely appreciated until the report of Barrett and Franklin [13] in 1949. (Adapted from Ellis and Olsen [16], by permission of W.B. Saunders Company.)

tance was mild and easily overcome, allowing the probe to be advanced to the stomach.

Fluoroscopic examination of the esophagus was performed using a contrast medium described as a thin suspension of bismuth. Flow of contrast medium was obstructed at the cardia. Further filling of the esophagus to the level of the tracheal bifurcation revealed that the esophagus was abnormally dilated and its lower end tapered, giving the appearance of an inverted wine bottle. There was a fine stream of thin contrast medium flowing into the stomach. On a subsequent occasion, a thickened mixture of contrast medium was used, but none of this passed through the cardia during lengthy observation. Heller's diagnosis was chronic "cardiospasm" (achalasia) with fusiform enlargement of the esophagus. There seems little doubt that his diagnosis was accurate.

Heller's Operation—The Prepared Mind

Heller's original report [2] revealed that he was most acutely aware of the then-recent publication by Heyrovsky [9] that described successes with side-to-side anastomotic esophagogastrostomy (aimed at bypassing the spastic lower esophageal sphincter) (see Fig 3). It is clear that Heller took his patient to the operating room with the full intent of performing the Heyrovsky procedure. He was somewhat apologetic about not at least attempting preoperative dilations or intraoperative transgastric retrograde dilation of the cardia, introduced by von Mikulicz [25] in 1903, but dismissed all such thoughts as inappropriate for his patient.

However, as Heller related in his 1914 article [2], during the course of the operation, as a result of certain observations, he abandoned his previous plan and performed an operation that was quite different, namely a transabdominal double (anterior and posterior) vertical extramucosal esophagomyotomy. Contrary to the apparent impulsive nature of this decision, Heller was quick to point out that the concept for such an operation was not new with him. Indeed, Gottstein [26] had expressed the opinion in 1901 that a procedure similar to pyloromyotomy could be performed at the cardia. This was entirely a theoretical suggestion, and until Heller, no one had performed an esophagomyotomy. Actually, Gottstein had additionally proposed closing the vertical myotomy transversely and extramucosally with stitches to protect the exposed mucosa from perforation, a suggestion Heller could not justify at operation and did not follow.

The operation was performed on Monday April 14, 1913. With a subcostal incision, good exposure of the proximal portion of the stomach was gained. The peritoneum overlying the esophagogastric junction was incised, the distal esophagus was encircled, and a 10-cm section of distal esophagus was exposed. The terminal portion of the esophagus was seen to be greatly narrowed for a distance of about 3 cm above the cardia. In this contracted portion, the esophagus was approximately the diameter of a man's little finger, while the esophagus above was described as being as big around as "the chimney of a kerosene lamp" with much hypertrophy of the muscular wall. The situation appeared to be suited for the performance of an extramucosal myotomy similar to that done by Ramstedt for pylorospasm in infants [18].

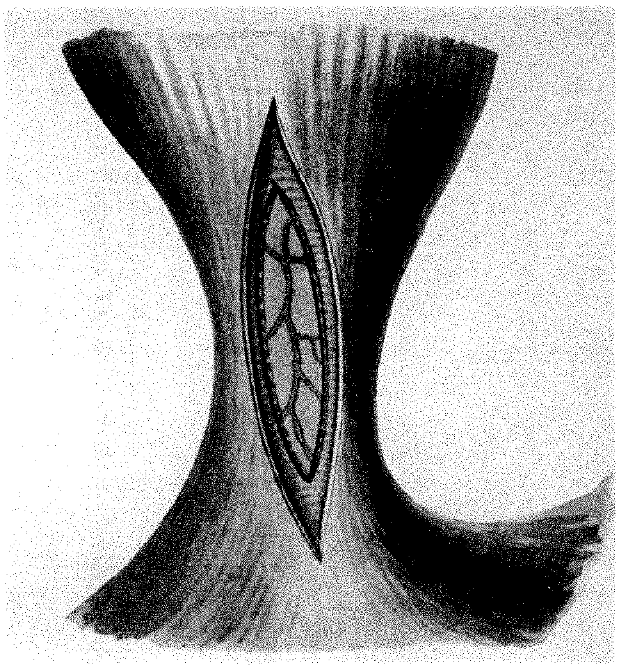

Fig 4. Reproduction of Heller's original drawing of his extramucosal esophagomyotomy (cardiaplastik) [2]. Note the complete division of longitudinal and circular muscle fibers and the vasculature of the exposed mucosa. Of special importance is the fact that the myotomy does not extend onto the stomach and was essentially an esophagomyotomy.

A longitudinal incision of 8.0 cm was made over the anterior aspect of the esophagus; superiorly it extended well upward onto the dilated and hypertrophied esophagus, and distally it ran only a short distance onto the stomach. Both his description and the accompanying drawing (Fig 4) suggest that Heller performed essentially an esophagomyotomy and not a totally sphincter-destroying esophagogastric myotomy.

The accuracy of Heller's observations continued as he carefully described splitting the fibers of the longitudinal muscle. As the fibers separated, the deeper circular muscle layers of the esophagus were revealed. In the more contracted part of the esophagus, the muscular coat was only 1 to 2 mm thick, whereas the more dilated and hypertrophied portion above the sphincter was many times thicker. Heller seemed pleased to find a vascular pattern over the exposed mucosa and was reassured that he did not have to close the myotomy as Gottstein [26] had theorized. Nonetheless, Heller tucked some omentum into this anterior myotomy with a stitch to maintain separation of the divided muscle.

Currently, the approach is to peel the muscularis off the mucosa for about 50% of the circumference of the esophageal mucosal tube to allow the mucosa to protrude through the myotomy site to prevent reapproximation of divided musculature with healing. This approach might have obviated Heller's next move, and that was to perform a second parallel myotomy on the posterior aspect of the distal portion of the esophagus. He deemed this necessary because the single anterior myotomy provided disappointing enlargement of the diameter of the narrowed segment. According to Heller, the tissues were friable and not suitable for safe suture anastomosis. Also to his apparent distress, there was no serosa covering the distal portion of the esophagus after mobilization.

Postoperative Course and Follow-up

In the immediate postoperative period, the patient had pneumonia in both lower lobes. This resolved completely without complication. From the very first day after the operation, the patient ate food in any form, even solids such as bread, meat, and potatoes. No further treatment was needed. The patient had completely normal subjective esophageal function. Heller warned against the use of any type of postoperative esophageal instrumentation because the lower esophageal mucosal tube was no longer protected by a mantle of muscle and might be vulnerable to perforation. This was a worry that was proved not to be a great risk.

The last known reexamination of Heller's patient was in 1921, nearly 8 years after the operation. According to Heller [3], the patient was in good health and nutrition at the age of 57 years. He could eat any kind of food provided it was consumed fairly slowly. Radiographic examination of the esophagus showed some narrowing at the cardia. In 1960, Heller stated that he had lost contact with the patient [3], and no further follow-up information was available after 1921.

Relevance to Current Practice

Aside from a bold and elegant theoretical and technical departure from the usual, Heller displayed a clear and decisive surgical mind when he [2] wrote: "apart from exposing the operative field, the essential problem in extramucosal myotomy is to completely sever the musculature without injuring the mucosa. That this can be done safely only under open operative conditions with direct visual control needs no further justification."

Modifications of Heller's *extramuköse cardiaplastik* have been legion, but the ambiguity of the term has led to considerable misunderstanding of just what is done in a "modified Heller procedure." The operation is something more than just performing a single anterolateral myotomy somewhere near the cardia. The technical questions are: where is the precise placement of the myotomy, and what is its linear extent? Heller was very clear on this point. Although he used an abdominal approach to the lower portion of the esophagus and cautioned his readers to divide completely all of the muscle fibers of the lower sphincter, it is apparent from both his text and illustration (see Fig 4) that the incision is to be limited to the distal part of the esophagus and not to be extended onto the stomach. Further, the myotomy was described as extending 8 cm proximal to the esophagogastric junction onto the more dilated and thickened portions of the esophagus. Thus, what Heller described was essentially an esophagomyotomy and not an esophagogastric myotomy [27]. This technical point becomes particularly important in light of the current controversy regarding the incidence of reflux after myotomy and the wisdom of incorporating an antireflux procedure when myotomy is done for achalasia [28].

Although this discussion probably cannot resolve that controversy, when a concerted effort is made to perform an esophagomyotomy in accordance with the principles outlined, late gastroesophageal reflux complications are extremely rare (3%) and substantial long-term relief of esophageal obstructive symptoms can be achieved—as in 90% of more than 5,000 surgically treated patients [28–30]. Accumulated evidence indicates that confining the myotomy to the esophagus and not extending it more than a few millimeters onto the stomach destroys the suprahiatal portion of the high-pressure zone associated with the lower esophageal sphincter in patients with achalasia, while retaining a vestige of the infrahiatal pressure barrier (Fig 5) [31, 32]. Extending an esophagomyotomy onto the stomach for 2 cm or more in patients with achalasia leads to complete destruction of the high-pressure zone and a 100% incidence of postoperative reflux [33].

So important is technique of the myotomy to clinical results that many guidelines have evolved to assist the surgeon in reaching a satisfactory end point for the distal extension of the myotomy. Most obvious is simply not to extend the myotomy onto the stomach, which can be further defined as not extending the incision beyond the point where the scant vasculature of the exposed esophageal mucosa merges with the rich vasculature or rete of exposed gastric submucosa. Thus, the myotomy can be extended a few millimeters onto the stomach for exposure of this gastric submucosal vascular pattern.

At times, this end point can be deceptively vague and imprecise. For this reason, other guidelines have been sought. As Heller [2] noted in his original publication, the lower esophageal sphincter in achalasia does not relax under general anesthesia. Thus, the surgeon might use intraoperative esophageal manometry as a guide to myotomy, in addition to the usual visual landmarks, with the goal of demonstrating that the myotomy has achieved at least a reduction of 50% in both the length and magnitude of the high-pressure zone (while at the same time preserving a remnant of the infrahiatal pressure barrier) [34]. Fundamental to current practice also is the separation of muscularis from mucosa in the region of the myotomy to allow the mucosa to bulge through the myotomy, thereby avoiding reapproximation of muscle fibers and restoring the obstructive sphincter. Generally, this separation is effected by freeing approximately 50% of the circumference of the esophageal mucosal tube of overlying muscularis.

Heller's esophagomyotomy concept has been extended to the management of other motility disturbances of the esophagus, namely, a miscellaneous group of conditions known collectively as diffuse spasm of the esophagus [35]. These include the "nutcracker" esophagus, the hypercontracting lower esophageal sphincter, the hypertensive lower esophageal sphincter, and segmental esophageal spasm. All of these conditions are associated with greatly increased esophageal motor activity that can be obliterated by myotomy, the site and extent of myotomy being

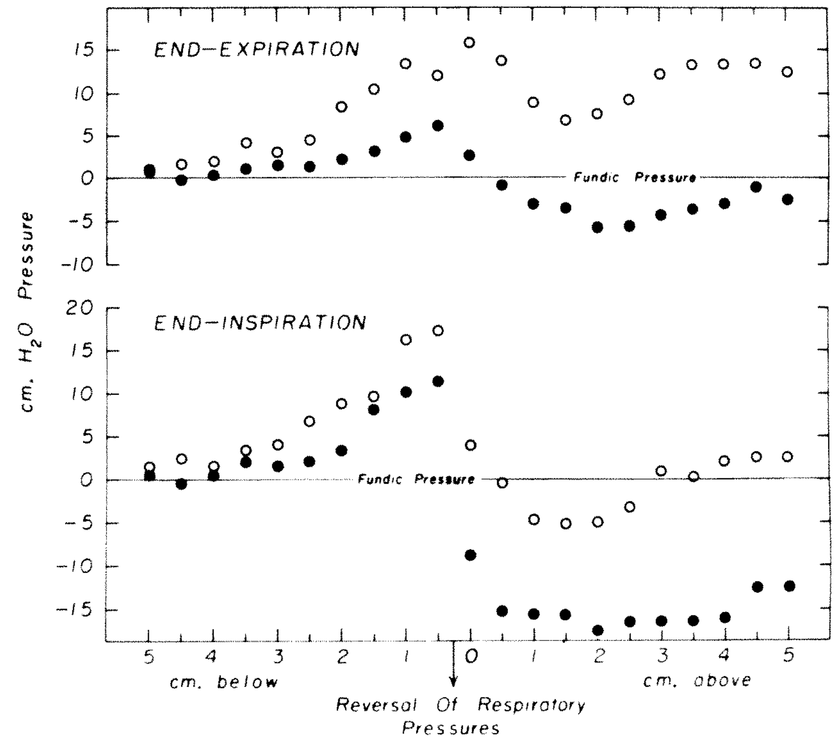

Fig 5. Mean resting pressures at gastroesophageal junction in 8 patients before (open circles) *and after* (solid circles) *esophagomyotomy. Note reduction in pressure after operation with partial retention of subhiatal segment. (Reprinted from Olsen et al [31] by permission of The C.V. Mosby Company.)*

influenced by specific manometric findings. The condition "vigorous achalasia" [36] is a variant of achalasia that responds to the standard modified Heller myotomy, which is generally extended cephalad to the level of the aortic arch to incorporate all areas involved in vigorous simultaneous contractions. Cricopharyngeal myotomy is yet another esophageal extension of Heller's contribution [37].

Although its recognition as a truly major advancement in the management of achalasia was slow, Heller's procedure has been refined and modified to meet various esophageal surgical needs, the most important of which continues to be the management of achalasia of the esophagus. The true value of myotomy over all of the other surgical procedures designed to destroy or bypass the nonrelaxing lower esophageal sphincter is that a remnant of sphincter is usually preserved while the obstructing portion is completely eliminated. The desired end point, unobstructed swallowing without gastroesophageal reflux, is thereby achieved.

Currently, the modified Heller myotomy is performed almost exclusively through a left thoracotomy (Fig 6). This decision is neither trivial nor arbitrary. When the operation is done through an abdominal incision, gastroesophageal reflux is almost twice as common as when it is done through a thoracic incision, regardless of whether an antireflux procedure is performed [28]. Disruption of supporting phrenoesophageal membranes may be more frequent when this route is used. More likely, however, the tendency to extend the myotomy onto the stomach produces the total sphincter-destroying esophagogastric myotomy.

Periodic rekindling of interest in forceful dilation of the lower esophageal sphincter using pneumatic or hydrostatic balloon dilation has great appeal to the surgical nihilist. When properly performed, however, esophagomyotomy remains the most effective therapy [28–30]. Although not all patients obtain the desired symptomatic relief, 90% do. Further, many thousands of consecutive

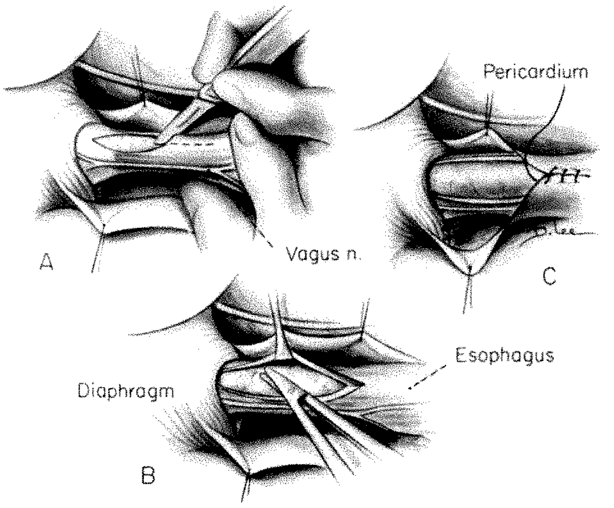

Fig 6. Technique of left transthoracic esophagomyotomy. (A) Beginning the incision. (B) Dissection of mucosa from muscularis. (C) Restoration of esophagogastric junction to intraabdominal position with suture narrowing of esophageal hiatus if necessary. (From Ellis FH Jr, Kiser JC, Schlegel JF, Earlam RJ, McVey JL, Olsen AM. Esophagomyotomy for esophageal achalasia: experimental, clinical, and manometric aspects. Ann Surg 1967;166:640–55. Reprinted by permission of J.B. Lippincott Company.)

operations have been performed with an operative mortality of only 2.8%. At the Mayo Clinic, more than 600 consecutive operations have been performed without a postoperative death, a record that not even the most enthusiastic advocate of balloon dilation can cite. As Heller [2] wrote, "that this can be done safely only under open operative conditions with direct visual control needs no further justification."

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Organ Procurement for Pulmonary Transplantation

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Selection of suitable donors is critical to the success of clinical pulmonary transplantation. Requirements for lung donors, management before explantation, and methods of preservation were reviewed for the 70 heart-lung, eight double-lung, and two single-lung transplantations performed at the University of Pittsburgh since 1982. Careful observation of trends of hyperoxygenation studies, chest roentgenograms, and Gram stain and culture results of tracheal secretions, as well as findings on bronchoscopy, can help identify which lungs not only have adequate function but are acceptable for transplantation. In spite of the rigid criteria used, 76% of tracheal cultures from donors deemed acceptable grew organisms. The presence of oropharyngeal flora has been shown to correlate with the development of early intrathoracic infections in the recipient. Prophylactic broad-spectrum antibiotic treatment of the donor is desirable to treat microbial contamination that could cause focal injury to the donor lung and predispose to infection in the recipient. Acceptance of less than ideal donors is ill-advised even though rejection of such donors conflicts with the current shortage of organs.

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Cardiopulmonary, single-lung, and double-lung transplantation have recently emerged as successful clinical procedures for end-stage lung diseases with or without associated heart failure [1–4].

The most critical factor limiting the growth of pulmonary transplantation is the shortage of suitable donors. It is estimated that only 10% to 15% of heart donors have lungs acceptable for transplantation [5]. In addition, the quality of the donor lungs is one of the most significant predictors of the clinical outcome of recipients [2, 5]. In view of the current shortage of heart-lung donors, a relatively larger number of single-lung and double-lung transplantations may be performed as current techniques allow for the separate extraction of the lungs and heart from the same donor [6]. Thus, some patients that once would have undergone combined heart and lung transplantation can now benefit from single-lung or double-lung transplants, while the heart can be used in a different recipient. Indications for the various forms of pulmonary transplantation that are currently employed at the University of Pittsburgh are in evolution as we increasingly recognize success with single-lung and double-lung transplantation.

The purpose of the present review is to describe the experience of the University of Pittsburgh in the procurement of organs for 70 combined heart and lung, eight double-lung, and two single-lung transplantations.

Donor Selection and Maintenance

The pulmonary donor must fulfill the common criteria for cardiac donors [7], as well the following requirements:

Age $\leq$ 45 years
Normal serial chest roentgenograms
Normal gas exchange ($PaO_2 \geq 400$ mm Hg on inspired oxygen fraction of 1.0)
Normal Gram stain of tracheal secretions (or culture if available)
Normal bronchoscopy
No history of primary pulmonary disease or active pulmonary infection
Appropriate size match with prospective recipient

The lung donor is evaluated by serial chest roentgenograms, repeated measurements of arterial oxygen tension (PaO_2), bacteriological examination of tracheal secretions, and bronchoscopy.

A clear chest roentgenogram is essential. Unfortunately, there has been substantial variability in the "local" interpretation of the chest roentgenograms. We have found that even mild abnormalities not considered serious are usually associated with parenchymal abnormalities that we believe would preclude successful transplantation. Therefore, we require that chest radiographs be repeated every eight hours and that a chest roentgenogram less than eight hours old be available at the time of procurement.

Determination of the PaO_2 on an inspired oxygen fraction of 1.0 and 5 cm H_2O of positive end-expiratory pressure should be performed at six- to eight-hour intervals. A PaO_2 below 400 mm Hg suggests serious ventilation/perfusion imbalance, often associated with anatomical abnormalities such as contusion, aspiration, infection, and atelectasis, which may not be obvious on the chest roentgenogram. Based on clinical experience, a progressive fall in repeated PaO_2 determinations is suggestive of an early, often radiographically silent, pneumonitis. The inspired oxygen fraction should be kept below 0.5 during

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the intervals between hyperoxygenation studies to lessen the risk of oxygen toxicity to the lung. It is preferred that peak inspiratory pressure be below 20 cm H_2O at a tidal volume of 15 mL/kg.

The tracheal aspirate should be cultured for bacteria and fungi, and a Gram stain should be performed. Culture results have been correlated with the onset of early thoracic infections in the recipient [8], but this information is usually not available before the explantation of the lungs due to time constraints. Gram stain of tracheal secretions is commonly used to evaluate the possibility of an ongoing subclinical pneumonia. As a general guideline, we feel that large numbers of bacteria or more than 15 neutrophils per high power field (×400) suggest the presence of bronchitis or pneumonitis. In addition, we consider the presence of heavy *Candida* species a contraindication to the use of lungs because of the high risk of fatal invasive candidiasis [9, 10].

Recently, we have initiated the routine use of bronchoscopy immediately before explantation, as infected sputum and aspirated material have been missed until late in the harvest procedure when the trachea is divided. Substantial aspiration of blood is a common finding in these patients. Early in our experience, postoperative atelectasis of the left lung prompted early bronchoscopy of a heart-lung recipient. The cause of the abnormality was found to be an endobronchial occlusion by a pickle slice presumed to be aspirated by the donor. Suboptimal suction of pulmonary secretions often may be due to small endotracheal tubes inserted expeditiously during resuscitation of the donor. The avoidance of infection by careful sterile technique in the management of urinary and intravascular catheters, tracheobronchial toilet, and wound care is essential. Frequent chest physiotherapy and deep repetitive endotracheal suctioning can be useful in preventing pulmonary sepsis.

The cause of death of donors in our series was closed head injury, resulting primarily from motor vehicle accidents, in 45% of cases and gun shot wound to the head in 40%. Other less frequent causes were intracerebral hemorrhage and primary brain tumors. After a motor vehicle accident, an apparent chest injury identified on direct visualization or on chest roentgenogram precludes lung donation. A unilateral pneumothorax or pulmonary contusion may lead to rejection of the double-lung transplant, but the opposite lung may be considered for single-lung transplantation. The circumstances of endotracheal intubation should be assessed as an emergency intubation performed at a site of a street accident carries a greater risk of contamination of the airways (aspiration of blood, gastric content) than the same procedure performed in a hospital setting.

In spite of the extensive screening protocol, it may be difficult to diagnose contusions and aspiration within 12 to 24 hours of brain death; therefore, we have rejected a number of these donors only after gross inspection ex vivo.

Occasionally, we have accepted suboptimal donors. In one instance, we used lungs from a donor with mild asthma who was on daily treatment with bronchodilators. The recipient died of infection in the early posttransplantation period. On another occasion we used lungs from a young donor who was a heavy cigarette smoker (more than two packs per day) with good results. Two other donors were being treated for active nonpulmonary infections: 1 with an intracerebral abscess and the other with tonsillitis. No complications were observed in the recipients, and we believe that patients with a recent history of adequately treated extrathoracic bacterial infections may be acceptable lung donors.

Although donors are matched with recipients for chest dimensions (circumferential, vertical, and horizontal thoracic measurements), we have no reservation in oversizing donors for recipients with obstructive pulmonary disease and increased thoracic volumes. To date we have not encountered serious difficulties with undersized donors even when the donor's lungs have failed to entirely fill the recipient's thorax and have left apical pneumothoraces. The latter is far preferable to the problems associated with compressive atelectasis of the lungs and cardiac tamponade due to the implantation of oversized lungs.

Blood group barriers are respected, and we do not consider blood group O as a universal donor as one of our A-blood-type recipients of a heart-lung transplant from an O-type donor developed hemolysis and acute renal failure. All donors are tested for hepatitis B, human immunodeficiency virus, syphilis, cytomegalovirus, and *Toxoplasma gondii.* A positive test for hepatitis B surface antigen or human immunodeficiency virus is a contraindication to procurement. It is ideal to avoid use of organs from cytomegalovirus-positive donors in seronegative recipients as such use carries an increased risk of cytomegalovirus disease [11, 12].

Among the complications of brain death, the most relevant to lung donation is neurogenic pulmonary edema. In an attempt to limit this complication, the pulmonary donor should be kept "dry" with a central venous pressure below 10 cm H_2O. This preference often conflicts with the desire of kidney transplant surgeons to maximize renal perfusion.

Lung Preservation

A number of preservation techniques are clinically available and provide for up to four or five hours of safe ischemia of the lung [13]. Early in our experience we procured 13 heart-lung transplants locally by means of bilateral thoracotomy with sternal transection, cardiopulmonary bypass, and profound hypothermia [14]. In 1984 normothermic autoperfusion of the heart-lung block [15] allowed effective distant procurement of 24 heart-lung blocks, but it was abandoned after 2 years in favor of the simpler static hypothermic preservation method [16] that has been used since then for the procurement of 33 heart-lung, eight double-lung, and two single-lung transplants. Prostaglandin E_1 (500 μg) is infused directly into the main pulmonary artery to vasodilate the pulmonary vascular bed. The heart is arrested with 1 L of crystalloid cardioplegic solution. This is followed by infusion (1,000 mL/min) by roller pump of a large volume (60 mL/kg) of modified Euro-Collins solution (10 mEq of $MgSO_4$ and 60 mL of 50% glucose per liter) at 4°C. Ischemic time for the pulmonary artery flush method of preservation was 182 ± 61.4 minutes (mean ± standard deviation), with a median of 192 minutes and a range of 40 to 279 minutes. With all of these methods, effective preservation of the heart-lung block has been achieved, but the predictability of good preservation has been erratic.

Other preservation methods for distant procurement that are currently used by other groups involved in lung transplantation include hypothermic cardiopulmonary bypass [17] and the use of blood-based hypothermic pulmonary flush [18]. The reintroduction of cardiopulmonary bypass for distant procurement has been made possible due to the development of lightweight portable pumps that can be easily transported in a small aircraft or an ambulance. Furthermore, cardiopulmonary bypass allows facilitated dissection of the mediastinum, obviates the need for close hemodynamic monitoring, and reduces the risk of hemodynamic collapse. This technique has resulted in adequate preservation in limited applications up to four hours both experimentally and clinically [19].

Postoperative Function

With static preservation the alveolar-arterial oxygen pressure gradient for a group of 11 consecutive heart-lung transplants was 304 ± 135 mm Hg (mean ± standard error) in the operating room on pure oxygen; 272 ± 167 mm Hg on the day of operation; and 180 ± 139 mm Hg on postoperative day 1, 161 ± 111 mm Hg on day 2, and 154 ± 108 mm Hg on day 3 on an inspired oxygen fraction of 0.4, at which level it remained until day 7 [Griffith BP, unpublished data]. These results did not differ from those obtained by autoperfusion [15].

Primary graft failure, defined as immediate deterioration of pulmonary function after successful revascularization of the transplanted lungs, has been observed in 5 patients (5/80, 6.2%). Three of these cases occurred in heart-lung transplants and two in double-lung transplants. The methods of preservation were hypothermic cardiopulmonary bypass (1), normothermic autoperfusion (1), and hypothermic pulmonary flush (3). Two cases appeared to be related to anoxic damage of the lungs during the period of preservation, as documented by histological signs of ischemic injury. The cause of primary graft failure in the three other instances has not been determined, but does not appear to be related either to sepsis or endotoxemia [20] in the donor or to hyperacute rejection, as determined by immunofluorescence and immunoperoxidase stains for complement, immunoglobulins G, M, and A, and fibrinogen.

In our last 37 heart-lung transplantations, donor trachea have been cultured and 76% of these cultures grew organisms [8]. All these lung donors fulfilled the criteria for acceptance and were deemed free from active infection and adequate for transplantation. The most common organism isolated from donor tracheal cultures was coagulase-positive *Staphylococcus* in 50% of all positive cultures (14/28). Oropharyngeal flora (defined as the presence of two or more of the following organisms: *Neisseria flava, Streptococcus viridans*, diphtheroids, microaerophilic *Streptococcus*, and coagulase-negative *Staphylococcus*) was identified in 46% of all positive donor tracheal cultures (13/28), suggesting aspiration of oropharyngeal secretions. Presence of mouth flora was associated with the development of early intrathoracic infections in the recipient ($p = 0.004$, Fisher's exact test, two-sided), whereas positive culture results for other organisms did not correlate with the onset of early intrathoracic infections. The duration of donor ventilation was 2.3 ± 1.7 days (mean ± standard deviation), and prolonged donor intubation was not associated with higher risk of early infections in the recipients. Seventy-five percent of infections occurring within 2 weeks of transplantation were bacterial pneumonias with a mortality of 50% (6/12). A heavy growth of *Candida* species in the donor tracheal cultures was associated with fatal thoracic infections in three of four occasions.

The organisms isolated from the donor tracheal cultures were different from the pathogens causing early intrathoracic infections in the recipients, with the exception of 2 cases in which donor cultures grew heavy *Candida* species. In these 2 patients, fatal invasive candidiasis developed with the same species of *Candida* that had been isolated from the donor (*Candida albicans* in 1 case and *Candida stellatoidea* in the other) [9]. These findings, together with the high rate of invasive fungal infections (23%) [21], have prompted the routine use of low-dose systemic amphotericin B (0.3 mg/kg/day) for the first 2 weeks after transplantation, unless unacceptable renal toxicity (serum creatinine level greater than 2.0 mg/100 mL) develops.

Future Directions

The major factor limiting the growth of pulmonary transplant programs worldwide is the scarcity of suitable lung donors [5, 12, 22]. This is mainly caused by the high incidence of pulmonary complications (infection, atelectasis, pulmonary edema) in potential donors.

The lung occupies a unique position in the body in that it is in direct contact with the external environment, and this leads to a high rate of microbial contamination and infection. This is confirmed by the high percentage of positive cultures of donor trachea [5, 8]. Moreover, when oropharyngeal flora was isolated from the donor trachea, the recipients were more likely to experience early intrathoracic infections compared with cases in which mouth flora was not isolated. As the organisms causing infection in the recipients are usually different from those isolated from the donor tracheal cultures, we believe the most likely mechanism is that contamination of the donor lung causes a focus of lung injury that sets the stage for superinfection in the recipient. This hypothesis have been verified in our laboratory in a canine model of donor-transmitted pneumonia, as we demonstrated that an inoculum of 10^4 colony-forming units of *Streptococcus pneumoniae*, which did not lead to pneumonia in normal dogs, led to widespread pneumonia in recipients of donor lungs that received the same inoculum 24 hours before transplantation [23]. We were also able to show that aerosol and intravenous antibiotic treatment of contaminated donor lungs prevented the development of pneumonia in the animals that received transplants [24]. As it is quite difficult to identify clinical donors at risk with a subclinical pneumonia, and because results of donor tracheal cultures are not available before explantation of lungs, we believe that all donors should be treated with broad-spectrum antibiotics. We feel that a combination such as ceftazidime (to provide gram-negative coverage) and clindamycin (to cover staphylococci, other gram-positive isolates, anaerobes, and *Mycoplasma hominis*) should be given to all lung donors as soon as they are recruited and should be continued until the time of explantation. The results of Gram stain and culture of tracheobronchial secretions will guide the adjustment of the initial prophylactic treatment. The high rate of invasive candidiasis in our lung transplant population, and the 2 cases of donor-transmitted fatal aortic candidiasis [9], prompted us to avoid use of donors if heavy *Candida* species were present in the tracheal secretions, and to institute a prophylactic low-dose treatment of all lung recipients with amphotericin B. Since adoption of this approach, no infection with *Candida* has been encountered in the last 15 lung recipients.

The importance of following trends in PaO_2 on pure oxygen is emphasized, as we feel a decline is the earliest indicator of undetected pneumonitis. We are suspicious of lungs that show a progressive fall in PaO_2, even if the PaO_2 remains greater than 300 mm Hg and a recent chest radiograph is clear. Findings at bronchoscopy and at gross examination are often the determining factor in such cases.

We have achieved adequate preservation with core cooling using cardiopulmonary bypass, normothermic autoperfusion, and hypothermic pulmonary flush techniques. However, we prefer hypothermic flush for its simplicity and effectiveness up to four or five hours of ischemia. Current research in organ preservation indicates that oxygen-derived free radicals are implicated in the microvascular and parenchymal cell injury associated with reperfusion of ischemic tissues [25]. The enzyme xanthine oxidase, released during ischemia from xanthine dehydrogenase by a calcium-triggered mechanism, is thought to be the major source of free radicals [26], and its inhibition has been shown to be beneficial in canine heart-lung transplants reperfused after 24 hours of cold storage [27]. The lung is much more sensitive to the effect of oxygen free radicals than other solid organs [28], and free radical scavengers (superoxide dismutase, catalase) added to the flush and/or to the storage fluid [29] have been demonstrated to reduce lung ischemic and reperfusion damage. Large numbers of polymorphonuclear leukocytes have also been found to migrate into the lung during injury because of oxidant stress [30]. Neutropenic animals that cannot increase lung leukocytes during hyperoxia have been shown to exhibit enhanced protection from oxygen toxicity [31]. Neutrophil depletion afforded protection against reperfusion-induced microvascular injury in experimental lung transplantation [32]. These promising experimental methods in long-term preservation of the lung may allow for prolonged ischemic times and may improve immediate graft function, but have not been applied clinically.

Selection of appropriate donors is crucial to success of pulmonary transplantation. Our present protocols are designed to determine which lungs have adequate function and to minimize the chances of development of infections in the recipients, as infection is the major cause of morbidity and mortality in lung transplantation.

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CORRESPONDENCE

Coronary–Subclavian Steal After Coronary Artery Bypass Grafting Using the Internal Mammary Artery

To the Editor:

In a recent issue, Olsen and colleagues [1] described the syndrome of coronary–subclavian steal through an internal mammary artery graft after coronary artery bypass grafting. They also collected 8 cases from the world literature.

In May 1985, my colleagues and I at De Stadsmaten Hospital, Enschede, the Netherlands, encountered such a syndrome in a 53-year-old woman. She underwent coronary artery bypass grafting in September 1984 because of worsening angina pectoris. The left internal mammary artery was used to revascularize the distal left anterior descending coronary artery and a saphenous vein graft was placed to the distal right coronary artery. In January 1985 the patient was rehospitalized with onset of recurrent angina after having been asymptomatic for some weeks. Digital subtraction angiogram showed a patency of the left IMA and saphenous vein graft. However, total occlusion was found at the origin of the left subclavian artery. To bypass this occlusion a left carotid–subclavian artery bypass [2] was performed (May 1985) and up to the present the patient is symptom free.

As the internal mammary artery is now an essential element in myocardial revascularization, it is our opinion, in agreement with that of Olsen and colleagues, that preoperative measurement of a possible difference in upper extremity blood pressure is a simple and effective means to screen for clinically significant stenosis of the subclavian artery. It is our policy to perform a digital subtraction angiogram or an angiography of the aortic arch-carotic-subclavian system only when there are signs of obstruction based on history and physical examination. If there are signs of obstruction the use of the ipsilateral internal mammary artery as a free graft, if possible the contralateral internal mammary artery, or a venous graft are alternatives in these patients.

This case mentioned here has been published in the Dutch *Hart Bulletin* [3], so up to the present 10 cases of this condition have been described.

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Reply

To the Editor:

Certainly more cases of coronary–subclavian steal through an internal mammary artery graft after coronary artery bypass grafting will become evident as manifestations of this syndrome are systematically searched for in patients who have undergone myocardial revascularization. Hopefully, with careful and thorough evaluation of patients before coronary bypass grafting, those with clinically significant subclavian stenosis can be properly identified and alternative conduits can be used. Dr Brouwer points out another excellent modality for diagnosing clinically significant subclavian artery stenosis, that of digital subtraction angiography.

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Autotransfusion of Shed Mediastinal Blood

To the Editor:

A recent paper by Griffith and associates [1] advocates the use of a cell washing device before autotransfusion of shed mediastinal blood. Recent work from our department, presented at the Surgical Research Society, has also focused on this phenomenon [2].

The study was part of a prospective randomized evaluation of intraoperative salvage autotransfusion using either the Solcotrans system or bank blood controls in elective aortic reconstruction. The Solcotrans device is similar in principle to the Sorensen system, and does not employ cell washing; salvaged blood is reinfused through a 40-μm filter. Systemic heparinization (2 mg/kg, controlled with the activated clotting time) was used during blood salvage.

To exclude the contribution of primary fibrinogenolysis, a latex immunoagglutination test for D-dimer, which is specific for fibrin split products, was used (MAbCO Ltd, Brisbane, Australia). Assays were performed on paired samples from autotransfusion reservoirs and from patients preoperatively and at 0, 4, and 24 hours postoperatively.

Normal levels (less than 200 ng/mL) were found in all patients preoperatively, whereas 28 of 32 reservoirs had raised levels of D-dimer (median, 3,770 ng/mL; range, 400 to 24,000 ng/mL). These levels are seen in high-grade disseminated intravascular coagulopathy. After reinfusion of salvaged blood, however, only 4 patients had elevated levels of D-dimer, not exceeding 500 ng/mL. No patients had raised levels of D-dimer 24 hours postoperatively. No evidence of coagulopathy was seen, and in particular there was no bleeding tendency, as evidenced by normal template bleeding times (Simplate-II, venous occlusion, single observer) and by wound drainage volume, which was less than that of bank blood controls.

The crux of both our study and that of Griffith and associates is the distinction between the detection of fibrin(ogen) split products in the autotransfusate or recipient and a clinical coagulopathy. Fibrinolytic products in chest drainage fluid derive from the action of tissue plasminogen activator secreted by the pleura, but coagulopathy has not been a problem with either the Sorensen or Solcotrans systems. Although high levels of fibrin degradation products have been shown to interfere with platelet function, bleeding times were normal in our study.

In established disseminated intravascular coagulopathy, the dynamic balance between coagulation and clot lysis is tipped in favor of coagulation, with a secondary activation of fibrinolysis, and a vicious circle is established. In contrast, in postoperative patients, there is a shutdown of fibrinolysis, with high levels of tissue plasminogen activator inhibitors [3]. Any plasmin infused during autotransfusion is presumably inactivated by circulating antiplasmins.

In summary, it would appear that our work is in agreement with that of the San Diego group, but we feel that the available evidence does not support the use of cell washing of shed

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mediastinal blood before reinfusion, which would entail extra cost and operator time.

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Reply
To the Editor:

I am pleased by Dr Thompson's discussion of our paper concerning the effects of infusion of fibrin split products from shed mediastinal blood collected from postoperative cardiac surgery patients. He adds healthy fuel to the fire of a yet unsettled debate over whether or not reinfusion of shed mediastinal blood has harmful effects. However, although he is correct in stating that the Solcotrans and Sorensen collection devices are fundamentally similar, unfortunately the conditions of his experiment are not the same as those in our experiment.

Thompson and associates collected systemically heparinized blood (2 mg/kg) from elective aortic reconstructions and tested the postinfusion data at 4 and 24 hours. On the other hand, we collected blood that was not heparinized, and tested the patient for fibrin split product levels one-half hour after infusion. These markedly discrepant time periods already make any comparative conclusions difficult, even if the blood in the study by Thompson and associates was not anticoagulated.

Thompson and associates measured levels of D-dimer fragments in the shed blood before reinfusion and found median levels of 3,770 ng/mL. In his letter, Dr Thompson states that only 4 patients had elevated serum levels of D-dimers after infusion but fails to state the volume of the infusate. This is a crucial piece of data, as a small infusate volume, regardless of the elevated D-dimer levels, would produce a low serum concentration and thus perhaps account for the negative results. Also, his results are at variance with those of Marcovitz and co-workers [1], who found D-dimer levels were elevated in patients 15 minutes after the reinfusion of shed mediastinal blood. Lawrence-Brown and colleagues [2] have confirmed that the elevated D-dimer levels in salvaged blood can be washed to normal levels, which gives credence to our results.

Dr Thompson purports an absence of bleeding tendency after reinfusion of the shed Solcotrans blood and, because of this, that washing is not justified. Unfortunately, Thompson and associates chose to use the template bleeding time and the volume of wound drainage to justify this conclusion. Bleeding times have little correlation with postoperative bleeding, especially in cardiac surgical patients [3], and there is generally sufficient variability in the postoperative blood loss of surgical patients to preclude showing significant differences between experimental conditions using this variable alone.

My colleagues and I applaud the attempts of Thompson and associates to advance knowledge on the hematologic effects of autotransfusion of shed blood but cannot accept their conclusions as applying to our experiment. The collected product, the timing of data collection, and the measurement tools are not similar to our experiment. When infusing large quantities of fibrin split products, there may still be unmeasurable negative effects that can be reduced by washing. In addition, the effects of infusing high levels of activated complement (which can be reduced by washing) are also unknown at the current time.

Continued experimentation should be encouraged to assure administering to the patient the highest quality autotransfusion product with the lowest attainable risk.

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Impairment in Right Ventricular Performance After Left Ventricular Aneurysm Operation
To the Editor:

We read with interest the article of Mangschau and co-workers [1]. We have studied right ventricular performance after coronary artery bypass grafting in our center with the use of a multilumen pulmonary artery catheter modified with a fast-response thermistor (Edward Laboratories, Santa Ana, CA) for measuring thermodilution right ventricular ejection fraction [2]. Unlike that of Mangschau and co-workers, our study included 34 patients scheduled for coronary artery bypass grafting without ventricular aneurysm during the first 48 hours of their postoperative course.

Despite the differences in methods, our results are similar to those of Mangschau and co-workers. In particular, we note the following.

1. We do not observe any correlation between right ventricular ejection fraction and pulmonary artery pressure. Other authors found the same results [3], probably because of the small dispersion of pulmonary artery pressure values and the alterations of the diastolic properties and compliance of the ischemic myocardium [4].
2. There is a significant decrease in right ventricular ejection fraction from 48% preoperatively to 37% four hours after coronary artery bypass grafting and 36% in the first postoperative day. This decrease is accompanied by a significant increase in heart rate and a significant decrease in right ventricular stroke work index during the same period. The right ventricular end-systolic volume index is unchanged, and we note a small decrease in right ventricular end-diastolic volume index.

3. There was no improvement in right ventricular performance after bypass grafting to the right coronary artery.

In conclusion, we are very happy about the results published in the work of Mangschau and associates, but we note the great difficulty in interpreting variations in right ventricular ejection fraction and variables of right ventricular performance influenced by preload, afterload, heart rate, and inotropism state. Moreover, as in the work by Mangschau and associates, we have been leaving the pericardium open [5, 6]. Finally, the effects of adhesions between the right ventricle and sternum are very difficult to estimate in the immediate postoperative course.

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Reply

To the Editor:

We appreciate the comments of Durand and co-workers. We are very pleased that their results in general are comparable with our data. However, several differences exist between these two studies in patient selection, morphology, methodology, and timing of investigations that make a direct comparison difficult.

Patients with a well-defined left ventricular aneurysm, like ours, probably have a more compromised left ventricle than patients scheduled for coronary artery bypass grafting. Interactions between the left and right ventricle are therefore more likely to occur in our study [1]. Moreover, our study was done a mean of 10 months after operation, in contrast to that of Durand and co-workers, who investigated their patients immediately after operation. This difference is important, because the right ventricle has an unique ability to recover during time [1]. However, the data from Durand and co-workers strengthen our belief that the impairment of the right ventricle is induced immediately after operation. The unsolved question is whether this impairment persists after uncomplicated bypass procedures or not.

We agree that great difficulty exists in interpreting variations in right ventricular ejection fraction, and that this may be influenced by several variables [1, 2]. To improve the validity of our data we therefore established the 95% confidence intervals for 20 healthy volunteers. Moreover, in our study preload and afterload values were unchanged after operation. However, we appreciate the results reported by Durand and co-workers using other methods.

Among the many possible reasons for right ventricular impairment, leaving the pericardium open and right ventricular adhesion to the sternum are among the most interesting [3, 4]. We agree, however, that the adverse effects of these factors are difficult to estimate. More accurate assessment of these effects should therefore be addressed in future studies.

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Retrotracheal Mediastinal Goiter With Contralateral Extension

To the Editor:

We would like to report a patient with a posterior mediastinal goiter that extended contralaterally, in whom the results of preoperative clinical and radiological evaluation were misleading.

A 65-year-old woman was seen with a large recurrent goiter. On clinical examination an enlarged right thyroid lobe that extended substernally was found. On the left side no mass could be palpated. Chest roentgenogram showed a right paratracheal posterior mediastinal mass, and the trachea was displaced ventrally on the lateral view (Fig 1). On computed tomographic scanning a voluminous goiter was found with substernal extension. The inferior pole of the right thyroid lobe expanded into the subcarinal region. The left lobe was judged to be only slightly enlarged.

The patient was referred for operation. The old cervical incision was opened. Many fibrous bands were found around the thyroid gland. The right lobe was slightly enlarged and extended only to the level of the manubrium in contrast to the findings on computed tomographic scanning. A right subtotal thyroidectomy was performed. The left lobe disappeared into the posterior mediastinum and its inferior pole could not be palpated. A full median sternotomy was then performed. The left lobe extended behind the trachea, which was compressed with torsion of its membranous part. The space between the ascending aorta and superior vena cava was opened. The carina was reached and the thyroid lobe spread for a distance of 3 cm on the right side of the trachea. Only then could the thyroid lobe be mobilized by manual dissection and delivered into the left cervical region. A

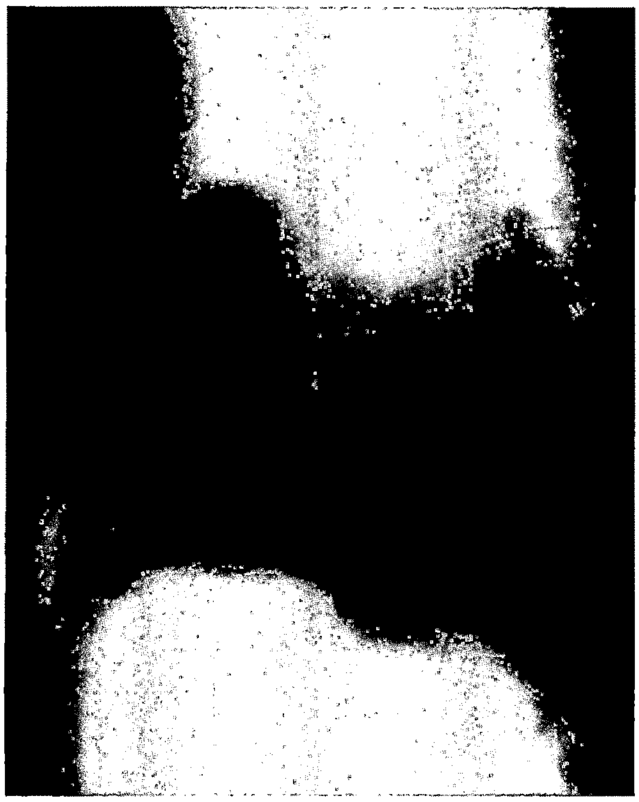

Fig 1. Lateral chest roetgenogram showing a mass in the posterior mediastinum displacing the trachea ventrally.

subtotal left thyroidectomy was performed. Pathological examination showed bilateral nodular hyperplasia.

Most secondary intrathoracic goiters occur in the anterior mediastinum, posterior extension being rarely found [1]. The case we present was a secondary goiter extending from the left cervical region into the posterior mediastinum. Probably the dense scar tissue resulting from the previous thyroidectomy forced the expanding thyroid into the loose posterior mediastinal tissue planes. The left lobe descended posteriorly between the trachea and the esophagus. Retroesophageal extension is more seldom found [2].

Probably because it provides only transverse slices of the mediastinum, preoperative computed tomographic scanning was unable to demonstrate this contralateral extension correctly. Thyroid scanning was also not helpful. Perhaps magnetic resonance imaging, which offers coronal and sagittal views, would have given the correct diagnosis.

Most secondary goiters can be mobilized from a cervical collar incision [3]. However, as the results of preoperative evaluation may be misleading, the surgeon should bear in mind that unusual locations are possible and that a full median sternotomy may be necessary to mobilize the thyroid entirely. In our patient it offered the additional advantage that both sides of the mediastinum could be reached and a one-stage thyroidectomy performed. A lateral thoracotomy as advocated by some [2, 4] would not have been helpful as the right mediastinal thyroid was in fact an extension of the left thyroid lobe, a finding that was not known preoperatively.

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KEY TO ABBREVIATIONS USED FOR ARTICLE CLASSIFICATION

* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence
CT = Collective Review
CV = Current Review
DI = Discussion
ED = Editorial
HW = How to Do It
KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

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* = Original Article
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CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence
CT = Collective Review
CV = Current Review
ED = Editorial
HW = How to Do It
KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

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  Evolution of surgical treatment of thoracic aortic aneurysms (Cooley) 1989;48:137–8,CL
  Gotthard Bülau and closed water-seal drainage for empyema, 1875–1891 (Meyer) 1989;48:597–9,CL
  Heller's contribution to the surgical treatment of achalasia of the esophagus (Payne) 1989;48:876–81,CL
  Lord Brock—the direct approach (Turley) 1989;48:736–7,CL
  Potassium-induced cardioplegia: evolution and present status (Gay) 1989;48:441–3,CL
Collagen
  Simultaneous use of microfibrillar collagen hemostat and blood saving devices in a canine kidney perfusion model (Niebauer, Oz, & others) 1989;48:523–7*
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  Blunt rupture of the myocardium (Pevec, Udekwu, & Peitzman) 1989;48:139–42,CT
  Pulmonary artery banding: analysis of a 25-year experience (Horowitz, Culpepper, & others) 1989;48:444–50,CT
Colon
  Reconstruction of the esophagus with the left colon (Huang, Sung, & others) 1989;48:660–4*
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  Use of colostomy bags for chest tube drainage (Kaul, Patwardhan, & Chaukar) 1989;48:456,CP; Reply (Velanovich & Adams) 1989;48:456,CP
Computed tomography
  Efficacy and benefit of mediastinal computed tomography as a selection method for mediastinoscopy (Thermann, Bluemm, & others) 1989;48:565–7*
Computer
  Use of artificial intelligence for the preoperative diagnosis of pulmonary lesions (Edwards, Schaefer, & others) 1989;48:556–9*

Conduits

- Construction of an aortic homograft conduit for right ventricle to pulmonary artery continuity (Hoots & Watson) 1989;48:731–2,HW
- Modified Danielson technique for recurrent aortic valve endocarditis (Dreyfus, Jebara, & others) 1989;48:725–7,HW
- Severe stenotic scar contracture of the Microvel Hemashield right-sided extracardiac conduit (Angelini, Witsenburg, & others) 1989;48:714–6,CR
- Use of conduits in surgical repair of congenital heart disease (Tuna & Danielson) 1989;48:143–4,KR

Congenital diseases

- Slide tracheoplasty for congenital funnel-shaped tracheal stenosis (Tsang, Murday, & others) 1989;48:632–5*

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- The arterial switch procedure for the neonate: coming of age (Laks) 1989;48:3–4,ED
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- Extracorporeal membrane oxygenation for circulatory support after repair of congenital heart defects (Weinhaus, Canter, & others) 1989;48:206–12*
- Lessons learned in pediatric heart transplantation (Trento, Griffith, & others) 1989;48:617–23*
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- Combined revascularization of coronary and femoral arteries: a proposed alternative (Suma, Sato, & others) 1989;48:434–6,CR
- Coronary arterial and sinusal anatomy in hearts with common arterial trunk (Suzuki, Ho, & others) 1989;48:792–7*
- Effects of atrial cardioplegia on the ischemic right ventricle after acute coronary artery occlusion and reperfusion (Diehl, Kaplan, & others) 1989;48:829–34*
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- Carotid and aortic arch endarterectomy using hypothermic arrest with coronary bypass (Nishida, Grooters, & others) 1989;48:865–6,CR
- Controversial aspects of coronary endarterectomy (Minale, Nikol, & others) 1989;48:235–41*
- Coronary artery bypass grafting in patients with ventricular fibrillation (Kron, Lerman, & others) 1989;48:85–9*
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- Coronary artery bypass in idiopathic thrombocytopenia without splenectomy (Thompson, Cohen, & others) 1989;48:721–2,CR
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  Heart-lung transplantation for pneumothorax in cystic fibrosis (Smyth, Scott, & others) 1989;48:744–5,CP; Reply (Spector) 1989;48:745,CP
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  Impairment in right ventricular performance after left ventricular aneurysm operation (Durand, Bastien, & others) 1989;48:888–9,CP; Reply (Mangschau, Geiran, & others) 1989;48:889,CP
  Internal banding for truncus arteriosus (Parnell) 1989;48:610,CP; Reply (Young) 1989;48:610,CP
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  Percutaneous venous cannula (Laub) 1989;48:610,CP
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  Hematological complications with the St. Jude valve and reduced-dose Coumadin (DiSesa, Collins, & Cohn) 1989;48:280–3*
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  Serial changes in renal function in cardiac surgical patients (Weinstein, Rao, & others) 1989;48:72–6*
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  Percutaneous cricothyroidostomy (minitracheostomy) for bronchial toilet: results of therapeutic and prophylactic use (Au, Walker, & others) 1989;48:850–2*
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  Cardiac retransplantation in the cyclosporine era (Dein, Oyer, & others) 1989;48:350–5*
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  Thromboembolism from undersized Dacron grafts in the descending thoracic aorta (Verdant, Page, & Baillot) 1989;48:686–8*
Data base system
  It is time for a national cardiothoracic surgical data base (Clark) 1989;48:755–6,ED
De Vega's procedure
  De Vega's annuloplasty for acquired tricuspid disease: Early and late results in 110 patients (Abe, Tukamoto, & others) 1989;48:670–6*
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  Mitral annular calcification: a new technique for valve replacement (Vander Salm) 1989;48:437–9,HW
  Treatment of median sternotomy wound infection and sternal necrosis in an infant (Terranova & Crawford) 1989;48:122–3,CR
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  Can ventricular decompression be optimized? (Lee) 1989;48:454,CP; Reply (Pennington) 1989;48:454,CP
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  Primary neurilemoma of the diaphragm (McClenathan & Okada) 1989;48:126–8,CR
  Spontaneous hemothorax in a patient with hereditary multiple exostoses (Teijeira, Caril, & Younge) 1989;48:717–8,CR
Disc valves
  Thrombotic obstruction of disc valves: clinical recognition and surgical management (Kontos, Schaff, & others) 1989;48:60–5*
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  Gotthard Bülau and closed water-seal drainage for empyema, 1875–1891 (Meyer) 1989;48:597–9,CL
  How soon should drainage tubes be removed after cardiac operations? (Smulders, Wiepking, & others) 1989;48:540–3*
  Use of colostomy bags for chest tube drainage (Kaul, Patwardhan, & Chaukar) 1989;48:456,CP

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Ductus diverticulum aneurysm and coronary stenosis: repair using circulatory arrest (Baisden, Sand, & others) 1989;48:432–3,CR
Ductus arteriosus, patent
Surgical closure of the patent ductus arteriosus in the neonatal intensive care unit (Coster, Gorton, & others) 1989;48:386–9*

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Detection of multiple cardiac papillary fibroelastomas using transesophageal echocardiography (de Virgilio, Dubrow, & others) 1989;48:119–21,CR
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Intraoperative evaluation of atrioventricular septal defect repair by color flow mapping echocardiography (Canter, Sekarski, & others) 1989;48:544–50*
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Are oxygenators (airplanes, oil spills, pesticides) safe? (Peirce) 1989;48:467–8,ED
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Effect of intraoperative intervention on neurological outcome based on EEG monitoring during cardiopulmonary bypass (Arom, Cohen, & Strobl) 1989;48:476–83*
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Candida endocarditis with saddle embolism: a successful surgical intervention (Kawamoto, Nakano, & others) 1989;48:723–4,CR
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Embryonic versus adult myocardium: adenine nucleotide degradation during ischemia (Mask, Abd-Elfattah, & others) 1989;48:109–12*
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Surgical preparation impairs release of endothelium-derived relaxing factor from human saphenous vein (Angelini, Christie, & others) 1989;48:417–20*

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Evaluation of arterialized vein graft permeability with Evans blue dye and iodine 125-labeled albumin (Finck, Mashburn, & others) 1989;48:646–50*

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Extracorporeal membrane oxygenation for circulatory support after repair of congenital heart defects (Weinhaus, Canter, & others) 1989;48:206–12*

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Treatment of postoperative chylothorax with intrapleural fibrin glue (Akaogi, Mitsui, & others) 1989;48:116–8,CR

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Detection of multiple cardiac papillary fibroelastomas using transesophageal echocardiography (de Virgilio, Dubrow, & others) 1989;48:119–21,CR

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Prevention of arrhythmias by flecainide after noncardiac thoracic surgery (Borgeat, Biollaz, & others) 1989;48:232–4*

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Prevalence of reoperation for pathway obstruction after Fontan operation (Fernandex, Costa, & others) 1989;48:654–9*

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Gentamicin solution for mediastinal irrigation: systemic absorption, bactericidal activity, and toxicity (Kopel, Riemersma, & others) 1989;48:228–31*

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Herbert Sloan, MD

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THE ANNALS OF THORACIC SURGERY

Official Journal of The Society of Thoracic Surgeons and the Southern Thoracic Surgical Association

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Specialty Review in Thoracic Surgery, Chicago, Illinois—January 8–13, 1990

This course is sponsored by the Cook County Graduate School of Medicine and is accredited for approximately 47 hours in category 1 of the Physician's Recognition Award of the AMA. For information, contact the Registrar's office: within Illinois, 1 (800) 621-4649; outside Illinois, 1 (800) 621-4651.

New Trends in Aortic Valve Surgery (Auto, Allo, Xenografts and Reconstruction), Riyadh, Saudi Arabia—January 17–18, 1990

For information on this workshop, contact Department of Cardiovascular Diseases, King Faisal Specialist Hospital & Research Centre, PO Box 3354, Riyadh 11211, Saudi Arabia (facsimile: 966-1-441-4839).

Esophageal Disorders: Pathophysiology and Treatment, Maui, Hawaii—January 25–30, 1990

This program is sponsored in part by educational grants from Synectics Medical, Inc, and Smith Kline & French Laboratories, and is accredited for 24 hours in category 1 of the Physicians Recognition Award of the AMA. For information on this meeting, contact Department of Surgery, Creighton University, CME Program, 601 North 30th St, Omaha, NE 68131; or telephone (402) 280-4501 (facsimile: (402) 280-4593).

Twenty-seventh Annual Seminar—Coronary Atherosclerosis: New Diagnostic and Therapeutic Approaches, Orlando, Florida—January 28–February 1, 1990.

This seminar is sponsored by the Departments of Radiology and Cardiology, Mount Sinai Medical Center of Greater Miami, Miami Beach, FL, and is accredited for 20.5 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Lucy R. Kelley, Program Coordinator, Radiology Seminars, Inc, PO Box 143762, Coral Gables, FL 33114-3762; or telephone (305) 674-2810.

Twentieth Congress of the Pan-Pacific Surgical Association, Kona, Hawaii—February 11–16, 1990

For information on this meeting, contact Pan-Pacific Surgical Association, 733 Bishop St, Suite 1910, Honolulu, HI 96813.

Nineteenth Annual Meeting of the German Society for Thoracic, Cardiac, and Vascular Surgery, Bad Nauheim, Federal Republic of Germany—February 22–24, 1990

For information on this meeting, contact Hagen D. Schulte, MD, Professor of Surgery, Department of Thoracic and Cardiovascular Surgery, Heinrich-Heine-University, Moorenstrasse 5, D-4000 Dusseldorf, Federal Republic of Germany.

Twenty-ninth Annual Waring-Durrance Chest Conference, Aspen, Colorado—March 7–10, 1990

This meeting is sponsored by the Colorado Trudeau Society and the American Lung Association of Colorado. For information on this meeting, contact American Lung Association of Colorado, 1600 Race St, Denver, CO 80206.

Loma Linda International Conference on Pediatric Heart Transplantation, Palm Desert, California—March 11–14, 1990

For information on this meeting, contact Leonard Bailey, MD, Loma Linda International Heart Institute, Loma

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Linda University Medical Center, Loma Linda, CA 92350; or telephone (800) INTL-HEART.

Thirty-ninth Annual Meeting of the American College of Cardiology, New Orleans, Louisiana—March 18–22, 1990

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone 1 (800) 253-4636.

Annual Meeting of the International Society for Heart Transplantation, Fort Lauderdale, Florida—April 4–6, 1990

For information on this meeting, contact International Society for Heart Transplantation, 435 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-0828.

Aortic Surgery Symposium II, New York, New York—May 4–5, 1990

This symposium is sponsored by Meadox Medicals, Inc, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA and toward the New York Medical Association Certificate in Continuing Medical Education. For information, contact Beverly Farrier, Symposium Coordinator, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660.

Seventieth Annual Meeting of The American Association for Thoracic Surgery, Toronto, Ontario, Canada—May 7–9, 1990

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Third International Symposium on Echocardiography and Doppler in Cardiac Surgery, Vienna, Austria—May 9–12, 1990

For information on this meeting, contact G. Maurer, MD, or W. Mohl, MD, PhD, c/o Interconvention, A-1450 Vienna, Austria; or telephone +43/1/23 69/2642 (telex: 11 18 03; facsimile: +43/1/23 69/648).

Eleventh Annual Scientific Session of the North American Society of Pacing and Electrophysiology, San Diego, California—May 31–June 2, 1990

For information on this meeting, contact the North American Society of Pacing and Electrophysiology, 13 Eaton Court, Wellesley Hills, MA 02181; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

Eighth Annual Uniformed Services University of Health Sciences Cardiothoracic Surgery Symposium, Washington, DC—May 31–June 2, 1990

For information on this meeting, contact Fred H. Edwards, MD, Department of Thoracic Surgery, Walter Reed Army Medical Center, Washington, DC 20307-5001; or telephone (202) 576-1433.

The Society for Vascular Surgery/International Society for Cardiovascular Surgery, Los Angeles, California—June 4–6, 1990

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Sixteenth Annual Meeting of The Western Thoracic Surgical Association, Coronado, California—June 20–24, 1990

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Thirty-ninth Congress of the European Society for Cardiovascular Surgery, Budapest, Hungary—September 9–12, 1990

For information on this meeting, contact Budapest Convention Centre, H-1444, PO Box 233, Budapest XI, Jagello u 1-3, Hungary; or telephone (361) 852-507 (facsimile: (361) 665-636; telex: 22-7717).

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the ad. To respond to an anonymous ad, write to Box Number, *The Annals of Thoracic Surgery*, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Cardiothoracic surgeon needed for rapidly expanding program located in a coastal area of the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Respond with curriculum vitae and names of references.

Please respond to Box 102. 102G/C

Cardiothoracic and vascular surgeon, board eligible/certified with experience in adult cardiac thoracic and vascular surgery, wanted to join well established 2-surgeon practice located in South Florida. Send CV.

Please respond to Box 105. 105C/B

Cardiothoracic surgeon to join established group in northwest Indiana (very near Chicago). Excellent salary. Please respond with curriculum vitae.

Please respond to Box 104. 104I/L

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join a large cardiac surgery group in southern California. Must have a minimum of 1 year experience in pediatric and neonate cardiac surgery (including fellowship). Excellent fringe benefits. Respond with curriculum vitae.

Please respond to Box 144. 144E/D

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 172. 172G/A

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/A

Thoracic/general surgeon, BC/BE, to join board certified thoracic/generalist in a busy noncardiac thoracic and general practice. Present physician is the only thoracic surgeon in this community of 80,000 which has a service population of 250,000. Practice is ideally located just one hour from a major metropolitan area and only 30 minutes from an international airport. First year guarantee, incentive, malpractice, and health benefits provided.

Please respond to Box 201. 201J/L

Thoracic surgeon, board certified/board eligible, wanted to join board certified thoracic surgeon in a 35-physician private practice multispecialty group in a waterfront community on Tampa Bay. Very active general surgery, vascular, and general thoracic practice.

Please respond to Box 214. 214J/L

Thoracic surgeon. A 220-physician multispecialty prepaid group in Hawaii is seeking a qualified MD to accept the second thoracic surgery position. Must be experienced in adult cardiac, general thoracic, and peripheral vascular surgery. Excellent fringe benefits. Malpractice paid.

Please direct curriculum vitae to Hawaii Permanente Medical Group, Inc, 3288 Moanalua Rd, Honolulu, HI 96819. An equal opportunity employer. 204J/L

Cardiovascular and thoracic surgeon wanted to join busy practice in Fort Lauderdale, Florida. Recent US medical graduate preferred.

Please send CV to I. Tabry, MD, 2773 NE 37th Drive, Fort Lauderdale, FL 33308. (Telephone: (305) 561-9781 after 9:00 PM.) 208J/C

A busy cardiothoracic group in the NYC area is looking primarily for a thoracic surgeon with potential for cardiac surgery in the future. Excellent salary and benefits. Respond with curriculum vitae.

Please respond to Box 211. 211J/L

Cardiothoracic surgeon, board certified with 2 to 4 years experience in cardiac surgery, wanted to join a growing group of cardiac surgeons, cardiologists, and pulmonologists in the Pacific Northwest. General thoracic and vascular experience necessary. Research opportunities available, academic links possible. Salary leading to full partnership in 2 or 3 years; potential exists for leadership in major cardiac program.

Please send curriculum vitae to John R. Bauman, Administrator at The Thoracic Clinic, PC, 507 NE 47th Ave, Portland OR 97213-2282. 124C/L

Cardiothoracic surgeon, BE/BC, to join expanding practice of cardiac/thoracic surgery at 719-bed hospital (Rhode Island Hospital) and major affiliate of medical school. Excellent clinical and investigational facilities available with ample opportunities for clinical or basic science research. Private practice with academic affiliation, solid general surgical residency with active medical student participation. Salaried position leading to eventual partnership for right individual.

Send curriculum vitae to Arun K. Singh, MD, 110 Lockwood St, Providence, RI 02903; (401) 274-7546. 217K/A

Cardiothoracic/vascular surgeon, BE/BC, with primary interest in cardiac surgery, wanted immediately to replace member of established 6-physician surgical group practicing in Allentown, PA. Competitive salary/fringe benefits. Position leads to partnership for successful candidate. Please respond with CV, history of clinical experience, references, and salary requirements.

Please respond to Box 218. 218K/D

Cardiothoracic surgeon: Exceptional opportunity for an additional cardiothoracic surgeon to join an established, multispecialty group practice in the Tampa Bay area. Recent graduate preferred. Must be experienced in all areas of adult cardiac, thoracic, and peripheral vascular surgery. Training in valve repair, electrophysiologic surgery, and left ventricular assist devices desirable. Guaranteed first year salary leading to potential directorship.

For immediate consideration, rush CV in confidence c/o Recruitment Committee: Diagnostic Clinic, PO Box 2901, Largo, FL 34649. 219K/A

Cardiovascular and thoracic surgeon—Board certified with 5–10 years experience in cardiac surgery wanted to start new adult program in regional medical center in northwestern lower Michigan. This modern practice situation is located in renowned midwestern vacation area featuring sailing, skiing, hunting, fishing, and other outdoor recreational opportunities as well as excellent schools. Please send CV and references.

Please respond to Box 220. 220K/L

Cardiovascular surgeon wanted to join busy private practice in excellent Florida location. Practice includes all aspects of adult cardiovascular and thoracic surgery. Would prefer a recent graduate. Send curriculum vitae.

Please respond to Box 223. 223K/A

Cardiothoracic surgeon wanted—to join established cardiac and thoracic surgeon in the Southeast. Private practice, primarily adult cardiac and thoracic. BC/BE required. Please send curriculum vitae.

Please respond to Box 225. 225K/A

Cardiac-thoracic surgeon, needed for expanding program on west coast of Florida. Excellent opportunity, private practice, primarily adult cardiac including cardiac transplantation. BC/BE required. Please send CV.

Please respond to Box 226. 226K/D

Thoracic surgery residency: Newly approved residency in thoracic surgery to begin July 1990 at the University of Massachusetts Medical School/Hospital. Three-year program includes training in adult and pediatric cardiac and general thoracic surgery.

Interested candidates may contact Thomas J. Vander Salm, MD, Department of Surgery, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655. 228K/A

Texas Gulf Coast institution is planning a new state-of-the-art cardiovascular program. Seeking an aggressive, young, and experienced cardiovascular surgeon skilled in myocardial revascularization, valve repair and replacement, cardiac emergencies, and vascular surgery. Unique opportunity in a contemporary environment enjoying the full support of administration and established staff. Initial contact must include curriculum vitae and personal references.

Please respond to Box 230. 230KL

Pediatric cardiovascular surgeon wanted to join full-time academic staff. Applicants should be board eligible or certified in thoracic surgery with a special interest in pediatric cardiovascular surgery and research. Additional transplant experience would be favorable.

Applicants should respond with curriculum vitae to Frank M. Midgley, MD, Chairman, Cardiovascular Surgery, Children's Hospital National Medical Center, 111 Michigan Avenue, NW, Washington, DC 20010; telephone: (202) 745-2137. 234KL

Cardiothoracic surgeon, BE/BC, wanted to join surgeon in active cardiac/thoracic/vascular surgical practice in northern Alabama community of 80,000 (300,000 population draw). Clinical practice in two community hospitals. Lake/mountain recreation facilities. Generous compensation, future partnership.

Please write Sara Fleming, Business Manager, 801 Gaines Ave, Suite 305, Gadsden, AL 35903; (205) 492-9360. 237L/E

Cardiothoracic surgeon wanted to join established practice in Long Island, NY, area. Recent graduate preferred. Please submit curriculum vitae and references.

Please respond to Box 238. 238L/B

Associate Director for Heart Transplantation is urgently needed for rapidly expanding heart transplantation program in California.

Please respond with curriculum vitae to the Pacific Cardiothoracic Surgery Group, 201 S Alvarado St, Suite 626, Los Angeles, CA 90057. 240L/K

Assistant surgeon—experienced in cardiothoracic surgery to first assist in the operating room. Active adult and pediatric cardiovascular surgery practice in southern California.

Please respond with curriculum vitae and outline of surgical experience to the Pacific Cardiothoracic Surgery Group, 201 S Alvarado St, Suite 626, Los Angeles, CA 90057. 241L/K

Cardiothoracic and vascular surgeon, BC/BE, recent graduate to join private practice of adult cardiac, pulmonary, and vascular surgery located in New Jersey. Please submit curriculum vitae.

Please respond to Box 243. 243L/C

Cardiothoracic surgeon needed to join established 2-man group in practice of adult cardiac, pulmonary, and vascular surgery. Must be board certified or eligible and graduate from a US medical school.

Please respond to R. W. Ruess, MD, 1834 Broadway, Cape Girardeau, MO 63701. 244L

Thoracic and cardiovascular surgeon, board eligible or board certified, wanted for an established, busy 2-man practice in Northeast. Must be familiar with all aspects of adult cardiac, thoracic, and peripheral vascular surgery including experience or training in laser-assisted balloon angioplasty. Salaried position leading to partnership. Submit curriculum vitae and references.

Please respond to Box 245. 245L/A

Cardiovascular thoracic surgeon wanted to join principal in metropolitan city on west coast of Florida. Must be ABTS certified or eligible and hold Florida license. Good opportunity for young, aggressive, energetic individual. Experience in cardiac transplantation an asset.

Please respond to Box 246. 246L

Cardiac surgeon, board certified or eligible, needed for a growing cardiac surgery program in the Southeast. Excellent opportunity for someone primarily interested in adult cardiac surgery.

Please respond to Box 248. 248L/B

Chief—Division of Cardiovascular and Thoracic Surgery: The University of Kentucky Medical Center, Lexington, KY, is seeking outstanding candidates as Chief of the Division of Cardiovascular and Thoracic Surgery. Applicants should have demonstrated clinical, teaching, research, and administrative skills.

Send curriculum vitae to Byron Young, MD, Johnston-Wright Chair of Surgery, University of Kentucky Medical Center, Lexington, KY 40536-0084. The University of Kentucky is an affirmative action/equal opportunity employer. 249L

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Please respond to Box 252. 252L/F

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Please respond to Box 254. 254L

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For information call Nell Franklin (704) 373-1500. 258L

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Please respond to Box 259. 259L

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Please respond to Box 260. 260L/A

Cardiothoracic surgeon: Cardiothoracic surgical faculty position available in the Division of Cardiothoracic Surgery, College of Medicine, The Pennsylvania State University, The Milton S. Hershey Medical Center. This is a full-time tenure track faculty position. Completion of residency training in an approved cardiothoracic surgery program is required. Candidate should be certified/awaiting certification by the American Board of Thoracic Surgery. Clinical experience in cardiac transplantation is desirable.

Please send inquiries and curriculum vitae to John A. Waldhausen, MD, Chairman, Department of Surgery, The Pennsylvania State University College of Medicine, PO Box 850, Hershey, PA 17033. The Pennsylvania State University is an affirmative action/equal opportunity employer. 261L

Faculty/academic position: The Division of Cardiothoracic Surgery, University of Washington, is seeking a BC/BE, adult cardiothoracic surgeon at the assistant/associate professor level. The individual will have a dual appointment at University Hospital and the VA and will share responsibility in establishing the VA clinical, educational, and research program. He/She should have a strong clinical background in cardiac and thoracic surgery and also have a demonstrated commitment to education and research. AA/EOE.

Send complete curriculum vitae to Edward D. Verrier, MD, Division of Cardiothoracic Surgery, RF-25, University of Washington, Seattle, WA 98195. 264L

Cardiovascular/thoracic surgeon, board certified or eligible; wanted to join busy private practice in the Midwest in adult cardiac, thoracic, and vascular surgery. Initial contact should include curriculum vitae, clinical experience, and names of references.

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Cardiothoracic surgeon, 33, American, university trained, wishes to relocate. ABS, BE for ABTS. Experience in IMA and AICD surgery. Seeks position in private practice or academics. Curriculum vitae and references upon request.

Please respond to Box 262. 262L/B

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Fellowship in thoracic and cardiovascular surgery at Baylor University Medical Center.

Contact Dr Harold C. Urschel, 3600 Gaston Avenue, Suite 1201, Dallas, TX 75246; telephone (214) 824-2503. 129G/L

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Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 222K/D

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Please respond with curriculum vitae to Adnan Cobanoglu, MD, The Oregon Health Sciences University, 3181 SW Sam Jackson Park Rd, Portland, OR 97201. 242L

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Please respond to Division of Pediatric Cardiac Surgery, The Heart Institute for Children, Christ Hospital and Medical Center, 4440 W 95th St, Oak Lawn, IL 60453; or telephone (312) 857-3029. 253L/D

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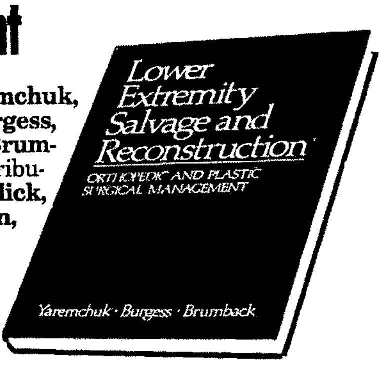
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By: **Michael J. Yaremchuk, MD, Andrew R. Burgess, MD,** and **Robert J. Brumback, MD,** with contributions by **Samuel S. Blick, MD, Paul N. Manson, MD,** and **Andrew J. Weiland, MD**

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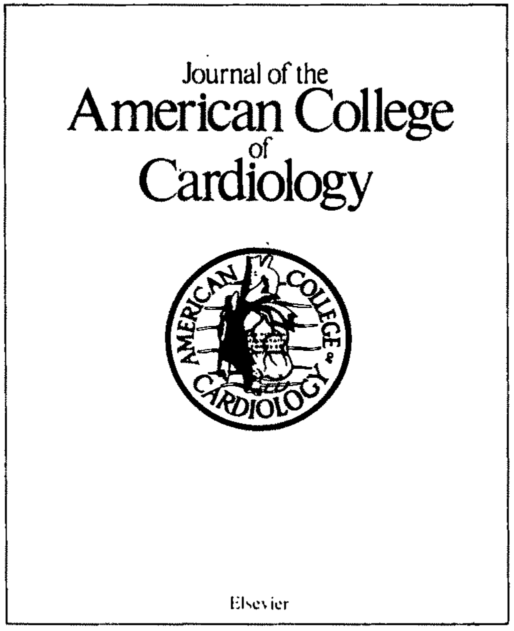

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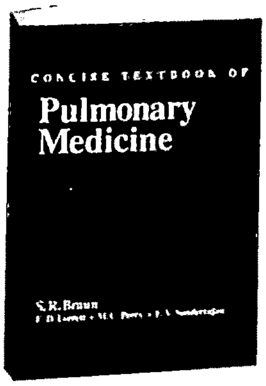

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